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TRUE SCIENCE IS THE KEY TO WISE PRACTICE

A TREATISE

ON

DISEASES OF THE LIVER

WITH AND WITHOUT JAUNDICE

WITH THE SPECIAL APPLICATION OF PHYSIOLOGICAL CHEMISTRY TO THEIR DIAGNOSIS AND TREATMENT

BY

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In Memoriam.

My original Monograph on Jaundice and Diseases of the Liver was dedicated in the following words :—

'TO

WILLIAM SHARPEY, M.D., LL.D., F.R.S.

Professor of Anatomy and Physiology in University College, London,

AS

A SMALL TOKEN OF A COLLEAGUE'S ESTEEM

FOR

A PROFOUND THINKER, A SOUND REASONER, AND A TRUE FRIEND.'

As the intervening eighteen years that elapsed between the day when I penned the above dedication, and my colleague's death, only tended still further to cement the bonds of our friendship, I gladly avail myself of this opportunity of re-endorsing the above sentiments, as a posthumous tribute to the memory of one who during a quarter of a century was ever ready to assist me by his advice, and always willing to guide me by his judgment. The intrinsic value of which boon it is impossible to over-estimate.

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PRELUDE.

For the sake of my own personal convenience I had written the MS. of this book according to the first stage of my proposed Progressive Scheme of National Spelling Reform. That is to say without encumbering its words (except personal names) with any redundant duplicated consonants. But a friend having pointed out that as all my medical books are extensively read by foreigners—indeed the last one, on the Urine, has not only been translated into French, but also into Italian—the omission of the duplicated consonants might add to their difficulty in perusing them, I have relinquished the idea of printing the book according to the proposed new method of improving the orthography of our language. I must candidly admit, however, that after having practised this improved method for five years, and tasted the sweets of its advantages, I have not been able to abandon the personal convenience accruing from it without a pang of regret.

Although I went fully into the question of National Spelling Reform in an article in the

'Medical Times and Gazette' of January 14, 1882, entitled 'National Spelling Reform in its Relationship to the Medical Profession, with a Scheme for its Consideration,' as I am most anxious to induce some of my younger medical brethren who may not only be sufficiently enlightened to appreciate the social value, but also have acumen enough to perceive the important medical bearings, of National Spelling Reform, to take up the subject, I shall here take the opportunity of saying a few more words upon a matter which I consider the whole medical profession should be alive to. The medical profession being the recognised public conservator of our national brain power. For its members as pure sanitarians, quite as much as humanitarians, ought to attach a much greater importance to the Spelling Reform movement than any other individual professional section of the community. In reading what I am now about to say on its strictly medical aspects the reader will perceive why I, as a practising physician, not only entered for a time the lists as a volunteer, but did my best to induce my medical brethren to follow my example.

That the mental well-being of millions of children is intimately connected with the question of Spelling Reform, is no mere chimerical assertion. It being well known to everyone who has studied the pernicious effects of the unnecessary mental strain which the inconsistencies of our defective system of orthography

entails upon every human brain, NOT IN THE POSSESSION OF AN INNATE SPELLING FACULTY. Everyone must have noticed how this is particularly the case with children, whose immature mental faculties are, according to the present system of school education, not alone strained to their utmost, but even far beyond it. The sad results of which are too well known to every reflecting member of the profession to necessitate my doing more than reminding them that the over-strain of school education often leads to permanent mental weakness, and in some cases to the development of active as well as incurable brain disease.

Although the pernicious effects of over mental strain in the acquirement of school education are abundantly apparent, their causes are not so easily recognised. So I purpose briefly to allude to some of them, in order to stir up among general practitioners an interest in the simplification of our system of teaching the young, particularly in so far as concerns orthography.

According to Dr. Gladstone, who is no mean authority on the subject, the chief obstacle in the way of primary education is our defective system of spelling. Which, he says, entails of itself no less than two additional, and consequently wasted, years of school study. Before I point out how this is readily accounted for by the inconsistencies, ay, I

may even unhesitatingly say the downright absurdities, which exist in the orthographical construction of English words, I desire to call special attention to a fact which is neither sufficiently known by schoolmasters and the general public, nor even sufficiently appreciated by medical men. Namely, that spelling power is an inborn gift, which cannot be in any case developed, though it can be in all cases increased, by cultivation.

What I mean by describing the spelling power as an inborn gift, is that the human brain possesses a spelling faculty, exactly as it possesses a calculating faculty, a musical faculty (called in common language a musical ear), the inborn gift of a steady hand or of a correct eye. And, as we all know, when any of these gifts of nature are constitutionally absent, it is absolutely impossible to drum them into a person, however clever he may otherwise be. So I now say, though it is a common, it is an absolutely erroneous notion to imagine that a person, however clever he may be, who is naturally constitutionally devoid of the spelling faculty, can ever, by any amount of cultivation, be made what is commonly called a good speller. No more is this possible than to make a person devoid of a musical ear a good musician. No doubt, by prolonged drumming and drudgery, he or she may be taught to play a faultless tune. So, by drumming and drudgery, a person

may be taught to spell by rote; but neither the one nor the other will ever be considered a proficient. In case any of my readers may doubt this fact as regards the spelling faculty, I beg to recall to their minds the cases of Sir Walter Scott and the Iron Duke, neither of whom to their dying day could ever trust themselves to write more than a friendly note without the aid of a dictionary. Indeed, so bad a speller was the highly gifted Sir Walter Scott, that the first five lines of his MS. of 'Peveril of the Peak' contained three misspelt words; and throughout the body of the MS.—as throughout all his other MSS.—misspellings occurred in superabundance. Some amusing anecdotes are told of the late Duke of Wellington's misspellings; but as most of my readers have no doubt heard of them, I need not take up time by relating any of them.

Now, as both of these men were undoubtedly not only possessed of a good education, but of more than ordinary mental abilities, their cases may be unconditionally accepted as furnishing us with conclusive proof that the spelling faculty is an inherent natural gift. But in case some may require still further evidence of this fact, I may remind them of the startling discovery which was made a few years ago—during the temporary existence of spelling bees—when it was found, not once, but often, that the best spellers existed among the semi-educated, and

the very worst of all among clergymen, lawyers, and doctors. Most of whom, as was seen, though perfectly *au fait* with common words, floundered and fell when asked to spell even what were supposed to be very simple words, merely because they happened not to be in everyday use. The cause of this is very simple. In many cases the orthography of English words is neither in accordance with orthographical general law nor with common reason. As every one knows, not a few, but actually hosts, of our English words are neither spelt according to sound nor sense, and, what is still more extraordinary, very many are actually spelt contrary to both. No wonder then that they not only puzzle the educated and reflecting mind, but are a stumbling-block in the way of the education of the intellectual as well as of the stupid, demanding a fearful amount of mental strain to master their inconsistencies and grapple with their defects. But what is the worst part of all is the fact that the mental strain happens to be required in the early years of life, when the brain is developing and immature, and consequently at the very period when it is least capable of bearing it. Should any reader be happily unconscious of the mental strain our defective orthography entails upon the brain, let him inquire of any foreigner what his opinion on the subject is, and he will soon learn the difficulties which even highly educated and powerfully intellected men

experience in acquiring a knowledge of English spelling. He will at the same time probably be quietly told that English spelling is, in the eyes of the educated foreigner, the very essence of sublime absurdity, and have many specimens immediately cited in proof of the statement. As for example, he may be told that we say 'kurnel' and spell it 'colonel.' We write 'bow,' and one time pronounce it as such, as when we speak of the bow of a boat or a salutation, and at another call it 'beau,' as when we allude to an instrument either to play a fiddle or to shoot with, notwithstanding that their origin and meaning, as well as their spelling, is the same. Further, it is not unlikely that the intellectual foreigner may blandly inquire upon what principles of grammar or sense we spell the *ough* in 'plough,' 'cough,' 'through,' 'enough,' and 'though,' seeing that they are all pronounced differently. What would the reader's answer be? I know not. So I would further ask, Why should we go on from day to day, from year to year, with this millstone hanging about our necks? Or perpetuate the mistake of forcing poor, helpless, and in many cases mentally delicate children into national schools, and insist on their learning absurdities, entailing upon their brains an amount of mental strain in many cases beyond their mental capacity?

It is really scandalous that so-called educated men continue to perpetuate tortures on immature intellects

by forcing them to acquire a knowledge of easily removable orthographical absurdities. The inconsistency of this mode of procedure, though not self-evident to the ordinary reader, is abundantly clear to the medically educated mind, who knows that it is not the ear that hears or the eye that sees, but the brain itself. The organs of hearing and seeing merely performing the respective mechanical parts of the process. Just as a speaking-trumpet or a pair of spectacles do. As the brain has no difficulty in distinguishing between a 'hair' and a 'hare' when presented to it in the same sound, how could it possibly have a difficulty when presented to it in the same spelling? We know for a fact that it has not. For, as just shown above, we spell four 'bows' all alike, and yet we instantly, from the context, know which is meant. Why then should we write 'you,' 'yew,' and 'ewe,' to symbolise the same sound, which is only correctly given by the single letter of the alphabet, *u*? Or why should we insist upon children spelling the sound of *ile* as 'isle' or 'aisle,' entailing a fearful amount of unnecessary perplexing mental drudgery? Is this, I ask, not a system of education alike derogatory to common sense, humanity, and sanitary law?

Were it only possible to get people who have paid no special attention to the subject, and, knowing next to nothing, understand still less, of the requirements of written language, to listen to those who have given

sufficient time to the mastering of its secrets, there would be but little difficulty in introducing a moderate and yet highly satisfactory scheme of spelling reform. No one wishes that every scheme of spelling reform, emanating from the brains of sanguine enthusiasts, should be accepted and acted upon. All that sensible men ask is the removal from the English language of the palpable and glaring orthographical inconsistencies which now needlessly infest it, and which are not only a stumbling-block in the way of primary education, but a fruitful source of mental injury; and it is on the latter ground that medical men, as the recognised sanitary advisers of the general public, ought not only to feel interested in, but assist in the advocacy of, the immediate introduction of an improved system of national spelling. Knowing as they all do that the wail of helpless children, quite as much as the cry of their bread-earning parents, is to be relieved from all unnecessary mental strain, both in the acquirement and in the diffusion of knowledge through the intervention of all kinds of mechanical agencies—whether they be pens, types, or telegraphs—and seeing that there is no object to be gained in wasting time, labour, money, and space on written or printed matter, why should we hesitate to improve our spelling, unless it be from the mere pseudo-sentimentality of 'letting things be'? On the same principle as the idiot boy refused to

have the nail which had lamed him removed from the heel of his boot, on account of his having got accustomed to it, and he would miss it if it were withdrawn! But even this pseudo-sentimental argument is untenable from the fact that language no more than anything else is, or ever was, stationary. Not only the spelling, but the very meaning of our words has changed, and is always changing. Thus, for example, the word 'silly' no longer means innocent, as it formerly did. Nor does 'candour' any longer mean whiteness, or 'virtue' bravery, or 'lewd' signify, as it originally did, unlearned. It is quite unnecessary for me to give examples of the strange mutations that have taken place in the course of the last three centuries in our forms of spelling, as every one must be abundantly familiar with them. Nor need I say a single word regarding the objection raised by those unacquainted with scientific etymology, that the reformation of our spelling would destroy the etymology of words, as every philologist knows that this idea arises simply from a deficiency of etymological knowledge.

I ought perhaps, however, to point out a fact of which but few of my readers are probably aware, namely, that the consideration of the desirability of reforming our spelling is not a mere 'whim of the day,' but is a subject which has occupied the attention of many reflecting minds ever since it was

first rocked in its infantile cradle by the learned hands of Dr. Gil—the tutor of Milton—no less than two hundred and fifty-nine years ago. Then it was but a feeble suckling, bound up in swaddling clothes; now it is a full-grown stripling, ready to perform its appropriate part in the affairs of life; and it only requires countenance and encouragement to become a potent lever in universal social development. It may, indeed, with perfect justice be said, that it will not do for this country to continue much longer to ignore its claims to consideration; for there is a destiny in language as in everything else, which awaits not the beck and call of frail human mortals. Blind although many may yet be to the fact, it is neverthless a discernible fact, that the time has gone by in the history of civilisation when a nation can complacently fold its hands and say, 'Let things be,' 'What did for our forefathers ought equally well to do for us.' Railways, telegraphs, and telephones have changed, and are changing, the whole relations of civilised society; and what was sufficient for the wants of our forefathers, two hundred or even one hundred years ago, is insufficient for ours now, and will be still less sufficient for the requirements of our successors. Mechanical language —the language which transmits from man to man knowledge by means of pencil, pen, type, or telegraph—requires simplification, and it is with the view of attaining to this end that I have here ven-

tured to address my medical brethren on the medical side of the question, as I have done in the 'Medical Times and Gazette' on its social aspects, and at the same time sketched out a 'Progressive Scheme of National Spelling Reform' for their consideration. A copy of which I shall be happy to send to any of my readers on application, feeling, as I do, that the social well-being of millions upon millions of the yet unborn must be materially influenced by the apathy or activity with which the question of Spelling Reform is handled in this the last quarter of the nineteenth century.

PREFACE.

In the year 1863 I published a monograph entitled 'Jaundice: its Pathology and Treatment, with the special Application of Physiological Chemistry to the Detection and Treatment of Diseases of the Liver and Pancreas;' and as, during the twenty years which have elapsed since I wrote it, my experience in the diagnosis and treatment of this particular class of affections has very greatly increased, and, naturally enough, my ideas regarding them have in an almost equal ratio expanded, I am tempted to again lay before my medical brethren the fruits of my further experience. In the hope that the immense advantages which the application of physiological chemistry affords the medical practitioner in the diagnosis and treatment of these diseases may speedily become, not alone more generally known, but at the same time far more generally adopted, than unfortunately they are at present.

This new treatise, which I have thought fit to entitle 'Diseases of the Liver, with and without Jaundice, with the special Application of Physiological

Chemistry to Diagnosis and Treatment,' although embodying within it the whole substance of my original monograph on Jaundice and Diseases of the Liver, bears no more resemblance to it than a mature adult does to the suckling from which he sprang. For although the scientific principles on which both works are founded are identical, the present is vastly greater than the former, both as regards its scope and materials. Containing as it does in a condensed form a large amount of clinical and scientific data that has never before been collected together by any author into one volume; while, in a great many instances, it gives a new rendering to old clinical facts by presenting them to the reader in the light of modern pathological science.

Moreover (for precisely the same reasons as I gave in the preface to my monograph on Jaundice for excluding from it the consideration of every question not having a direct bearing on the elucidation of the matter in hand), as I still think time is quite of as much value to the professional as it is to the mercantile man, I have endeavoured to condense my materials to the utmost without running the risk of endangering their perspicuity. Added to which, as this treatise has not been penned, either for the use of the tyro or the dilettante in medicine, but for that of my qualified brethren, I shall neither waste time by entering into detailed accounts of the

literature, nor give tedious, and probably at the same time profitless, discussions of the theories of the mechanism of jaundice in hepatic derangements, but limit myself entirely to a brief exposition of my own views. Taking care, however, in order that they may carry more weight with them in the eyes of the reader, to illustrate them freely with cases reported by independent observers both at home and abroad. While, in order again that the reader may be able to see for himself at a glance how many of the old-fashioned theories of the pathology of jaundice have been abandoned, as well as how many new ones have been espoused, I have put my views, in accordance with the facts and arguments expressed throughout the body of the volume, as in my monograph on Jaundice, into a concise and diagrammatic tabular form at p. 801.

I may further add that as the object of all theory, and the aim of all science, is to ensure wise practice, I desire to call special attention to that portion of the work devoted to the physiological chemistry of the excretions. Feeling, as I do, that we are entering upon the threshold of an important line of medical inquiry, which, sooner or later, will be followed by valuable practical results. I would also direct the special attention of my readers to the chapter devoted to treatment, as well as that at the end of the book entitled ‘ Hints on Diagnosis,’ being sanguine enough

to imagine that the adoption of the principles enunciated regarding the physical and chemical methods of diagnosing diseases of the liver, as well as of the modes of action and administration of the remedies usually employed in hepatic affections, may conduce to a more rational and successful method of treating them than has hitherto been employed. I even go so far as to hope that the result of the diagnosis and treatment, as shown in many of the cases cited, will not only justify the adoption of the principles on which they are founded, but also prove a strong incentive to others to follow the physiological chemical line of investigation I have striven to inculcate.

In some portions of the volume the statements may, perhaps, appear to be rather dogmatic; if so, I may remind the censorious reader that this has arisen from the circumstance of so many old dogmas and deeply rooted prejudices having to be combated. For I am quite as alive as he can possibly be to the fact that what one may regard as scientific truth is in no case incontrovertible certitude, and that the deductions of to-day in an advancing science like that of medicine may require material alteration when viewed in the light of the morrow. But I am still equally convinced of the fact, that if men fold their arms, and refrain from acting until every link in the chain of knowledge is made perfect, all progress will be arrested, and the day of certainty still further postponed.

Too long have the members of the so-called practical school of practitioners reversed the natural order of things, and commenced the study of medicine where they ought rather to have left it off. Too long have they striven, by studying pathology ere they were sufficiently acquainted with physiology, to place the pyramid of medical science on its apex instead of on its base; and this is, I believe, the reason why they have remained so long ignorant of the fundamental doctrine that the same laws which regulate health also regulate disease.

Nature does nothing on a small scale, and the more we study her the more we are forced to admire the uniformity and extensive applicability of her laws. If, for example, we pry into the ultimate structure of our bones, we find they receive their nutriment by a system of irrigation carried on through lakes and rivers (lacunæ and canaliculi); and if we examine the periosteum surrounding them, the ligaments attaching them, or the muscles covering them, we still find that, notwithstanding their diversity in structure and in use, the one system of irrigation pervades them all. We may even go a step further, and say that the same laws which govern the animal govern also the vegetable kingdom. For. the further science advances, the more apparent does it become, that not only the animal and vegetable, but even the organic and inorganic, form but one

world, being but mere modifications in the arrangement of the self-same elements, one and all being regulated by the same laws.

A knowledge of animal organisation, important though it be, is yet less indispensable to the physician than a knowledge of healthy function, for it is the latter which more especially elucidates the dark problems of life. It is the latter which proves the golden key to the comprehension of disease.

Although not even the most ardent admirers of medicine can say that it as yet merits the name of an exact science, this ought neither to destroy our hopes nor trammel our labours. With the stethoscope, microscope, test-tube, thermometer, and other physical means of diagnosis, a new era dawned upon our art; and now the members of the advanced school of thinkers which is rising up, and carrying chemistry into the domains of medicine, are but the pioneers of the revolution which is soon to follow. If we look back to what the exact sciences of to-day were in former times, we shall find they were much less perfect then than medicine is now. Astronomy and chemistry a century or two ago were but astrology and alchemy. If, then, we may draw a picture of the future from the progress of the past, we need have no hesitation in saying that chemistry rightly applied, and physiology justly interpreted, will, ere many generations pass away, reveal the deepest secrets of

diseased action ; and although scientific medicine will still be, and for ever remain, unable to banish death, it will nevertheless enable the properly trained scientific practitioner to follow with unerring certainty the various morbid changes occurring in the human frame, as well as probably at the same time permit him to mould their course to the advantage of suffering humanity.

25, Harley Street, W., 1882.

CHAPTER I.

GENERAL REMARKS ON THE STUDY OF DISEASES OF THE LIVER.

The practical experience I have acquired in the diagnosis and treatment of diseases of the liver since the publication of my monograph on Jaundice admits of my now asserting, without hesitation, that the old and still prevalent idea that hepatic diseases are, as a class, exceptionally difficult to diagnose, is totally at variance with truth; although I must at the same time admit that the pathology of at least some few of them is not only frequently puzzling, but occasionally obscure.

The errors which one every day sees committed in the differential diagnosis of diseases of the liver are not, I believe, due, as is commonly asserted, to the uncertainty of their symptomatology and the inherent difficulties attached to their signs—from the supposed peculiarly obscure nature of the morbid physical conditions themselves—but in a great measure to the defective means adopted by practitioners in attempting to unravel them.

While the physical means of diagnosing cardiac, pulmonary, and renal affections have gone on steadily improving during the last quarter of a century, the physical agents now employed by the vast majority of physicians in diagnosing hepatic affections are scarcely, if at all, one whit better than they were half a century ago.

And yet, as the sequel will prove, this is not due to an absence of means, but to a disinclination to take the trouble of employing them. At whose door does the blame lie? To this question I reply, in very many instances, at the door of the teachers of clinical medicine, not one, but many of whom give the students under their charge but little opportunity of becoming practically acquainted with the different forms and phases of the few hepatic cases in the wards of their respective hospitals. Hour after hour of the students' limited, and consequently valuable, time being expended by their teachers in the physical diagnosis and treatment of thoracic affections, to the almost total exclusion of liver derangements; just as if affections of the liver were things of mere secondary importance to those of the heart and lungs.[1] Perhaps the prominence generally

[1] However much it may seem to the contrary, I beg to observe that no assertion is made in this book, no matter however *bizarre* it may at first sight appear to the mind of the uninitiated, which I do not believe will bear the strictest investigation. Even this startling accusation of general professional ignorance, which has just been made, I could bring ample printed

given by the majority of clinical teachers to thoracic affections may partially arise from the fact that while they themselves excel in physical diagnosis, by means of auscultation and percussion, they are perhaps conscious that the inadequacy of their own physiological chemical training renders it almost impossible for them to grapple successfully with some of the complicated cases of hepatic disease. Experience, the most unmerciful of all teachers, having taught them that complicated cases of liver disease cannot be properly mastered without the aid of physiological chemistry. From the fact that diseases of the liver are not, like those of the heart and lungs, amenable to direct

evidence to support; as, for example, Professors Parkes and Longmore's statements given in the *Report on Medical Education of the British Medical Association*, dated Jan. 1881, from page 8 of which I extract the following:—

Professor Longmore, writing in November, 1879, on this subject, observes: 'We see at Netley how little qualified in practical professional matters some of the young men are at starting who have passed the examinations for their licenses to practise, in many instances with much credit. Out of a batch of twelve men, now at Netley, all above the average, not one could make a quantitative analysis of the urine, and only a few had a practical knowledge how to make a qualitative analysis. It is only in a few exceptional instances that I find a young surgeon coming to Netley acquainted with the manipulation of the ophthalmoscope, or knowing how to determine the refractive quality of an eye. As a general rule, it is absolutely necessary prescriptions should be supervised; and so on in numerous practical matters.' It is argued that the men who enter at Netley are hardly up to the average standard of practitioners; this is an error, but if true, these gentlemen have all received diplomas and are legally qualified practitioners, and it is further said—'We find precisely similar evidence bearing on this subject, repeated over and over again by general practitioners residing in every part of the United Kingdom.'

interrogation by means of the stethoscope and plexi-meter. Auscultation being in fact of no avail whatever, and percussion yielding nothing more than the meagre information derivable from the one solitary factor of bulk.

Physiological chemistry, on the other hand—as there will be ample occasion for seeing in the sequel—is the only talisman which can unravel the tangled skein of secrets which involves the vast majority of hepatic disorders.

In thus speaking of physiological chemistry, I do not desire it to be for a moment supposed that I imply by that term the mere paltry smattering of physiological chemical knowledge that is picked up by a man of average ability in the lecture-room during an ordinary course of medical study. For, on the contrary, that amount of physiological chemical knowledge will never suffice for the detection and differentiation of hepatic diseases, unless it be supplemented with some practical chemical manipulative skill. It would be just about as absurd for an ordinarily educated student to think that he is a physiological chemist, as for a retail druggist to imagine himself a scientific chemist because he possesses a spirit-lamp, a test-tube or two, and a set of chemical reagents, no matter however anxious he may be to make the public think so by having the word 'CHEMIST' placarded in large letters over

his shop-door.[1] Physiological chemistry is a distinct branch of science, which is not to be mastered in either a week, or a month, or a year, and he who desires to profit by its teachings at the bedside of his patient must be content to begin its study at the analytical table of a practical laboratory; for it is there alone that can be acquired the requisite amount of preliminary information to enable him to turn physiological chemistry to useful account in the sick-chamber.

If these, then, are true facts, how is it possible for one to feel in the least degree surprised that the

[1] In no country of Europe except England, not even in benighted Spain, where barber-surgeons exist until this very day, do men who retail medicines style themselves 'chemists.' A chemist is a man who works with chemicals, not a man who sells them. Far less a man who merely sells drugs, many of which are not even so much as in the common sense of the word chemicals at all. An infusion of gentian or a decoction of broom tops, for example, has no more right to the title of a chemical preparation than a cup of tea or a pot of coffee.

There are in England manufacturing druggists as well as a class of legally qualified druggists—who from having obtained the diploma of the Pharmaceutical Society have a perfect right to call themselves pharmaceutical chemists—but it would be quite as much a solecism, and consequently wrong, for them to drop the first half of their title and simply retain that of chemist above their shop-doors, as it would be for a dental surgeon to drop the word 'dental,' which is equally in the same sense indicative of his special calling, and simply retain that of surgeon on his door-plate. There is in reality the same philological difference between a manufacturing, dispensing, and pharmaceutical chemist, and a 'pure chemist,' as there is between a 'dental surgeon' and a 'surgeon.'

If then the sellers of medicines who do not possess the diploma of the Pharmaceutical Society dislike the name of druggist (about which there is nothing whatever derogatory), let them prefix the word 'dispensing,' which will unmistakably denote their avocation, and by so doing cease to sail under false colours by putting over their shop-doors the single word chemist.

present imperfectly chemically educated race of medical students, when transformed into legitimately licensed medical practitioners, on encountering a not self-evident case of diseased liver, at once find their diagnostic powers not only entirely inadequate for its solution, but at the same time feel themselves to be tossed hither and thither on a most uncomfortable sea of perplexity and danger as regards its treatment? Ay, still more, it may be equally truthfully said—and that too for a precisely similar set of reasons—that it not unfrequently happens that some of our most talented clinical teachers, as well as the cleverest of our consultants, of justly acknowledged skill in the diagnosis of disease affecting other organs of the body, are completely baffled in their attempts to unravel what they are forced in self-defence to designate 'obscure diseases of the hepatic organ.' Occasionally wandering, as will subsequently be shown, so far astray as to diagnose the case as one of cancer of the liver,

A word which has been already otherwise appropriated in every country of Europe, and carries with it a special and entirely different signification from that of a salesman of medicines in the mind of every well-educated man, be his nationality what it may. Indeed I see no reason why those gentlemen who have not only received a practical chemical education, but are at the same time Fellows of the Chemical Society, should object to prefix the word Dispensing. Just as many others do Analytical before the word Chemist, in order to indicate their special avocation. For even numbers of gentlemen having a right to the title of surgeon, from possessing the diploma of Member or even of Fellow of the Royal College of Surgeons, who practise Dentistry as a speciality, always, I believe, employ the distinguishing duplex title of Surgeon-Dentist.

when not even a trace of such morbid material exists in the organ, or even so much as in the system; and yet again failing to recognise the presence of a malignant form of this inevitably fatal liver affection until the patient is on the very brink of the grave. Such startling errors in diagnosis are by no means always traceable to the symptoms and signs of organic change having been present in the former and absent in the latter set of cases, but in general, either from the physician having omitted to adopt the proper means for the detection of the morbid condition. Or, if having made use of them, from his failing to recognise the true import of the revelation they have made to him. From his inability to correctly interpret and sufficiently appreciate their meanings, even when he has them before his eyes.

To the reader not much versed in the history of hepatic cases this may perhaps seem strong, if not even ungenerous, language for me to use. But every consultant who, like myself, has given special attention to the clinical history of liver cases, and consequently who is as it were behind the scenes, will, I feel sure, unhesitatingly endorse the remark. I know full well that although I may be the first to put such a statement in type, I am a long way from being the first that has entertained the sentiment. Indeed, should any hypercritical-minded reader feel inclined

to challenge its truthfulness, let him turn to the report, meagre and imperfect though it be, of the discussion which followed the reading of a paper on Choleocystotomy in a case of impacted gall-stone, read before the Medico-Chirurgical Society, which appeared in the medical weeklies of November 15, 1879, and after the perusal of the observations therein said to have fallen from the lips of some of the speakers on that occasion, I think he will cease to doubt either the justice or the propriety of the remarks, as well as cease to wonder that the medical 'art' has been, by cynical critics, stigmatised as an incomprehensible conglomeration of false facts, cemented together by unsupportable theories. Moreover, I will even dare to ask my hypercritical-minded reader if this can be regarded as astonishing when we look around us and see still in vogue, on all sides, the non-philosophical, non-physiological system of treating and speaking of mere symptoms, as if they were in reality diseases—that is to say, palpable physical morbid states.

Can any one be found bold enough, I will ask, to say that this remark is not specially true as regards liver cases, the therapeutics of which is but little better now than it was a century ago? For beyond the occasional introduction into prescriptions of one or another of the vegetable hepatic stimulants newly introduced from America, the sum and substance of

the vast majority of them neither differ in quality nor quantity from those in general use among English practitioners at the beginning of the present century—a fact not to be wondered at, seeing that many of the men at present occupying the posts of clinical teachers in our metropolitan medical schools lecture to their pupils on what they please to term mere 'Functional Derangements of the Liver,' and teach them to prescribe for this so-called species of hepatic disease as if the states of body they described were in themselves morbid physical conditions, instead of being, as they in reality are, mere concomitant symptoms of not unfrequently very widely differing morbid physical conditions of the hepatic organ. For just as pain is a symptom common to many diseases, so are jaundice, pipeclay-coloured stools, and saffron-tinted urine, signs common to some diametrically different forms of affections of the liver.

Yet, marvellous to relate, this very same class of otherwise enlightened clinical teachers, in spite of their following this pernicious system of teaching students 'how to treat functional hepatic diseases,' would repudiate with scorn the bare suspicion of being considered capable of prescribing for a mere 'palpitation of the heart,' a 'bronchial expectoration,' or a 'purulent urine,' under the title of functional diseases of the heart, lung, or kidney.

Nay, more, one and all of them would naturally

enough look with supreme contempt upon the mere idea of being thought capable of condescending to treat like a routine empiric what is called a mere symptom—such, for example, as a headache or stomach-ache—even if presented to the uninitiated minds of their confiding patients disguised from their cognisance in the grandiloquent Greek synonyms of cephalalgia and epigastric neuralgia. Yet, strange to say, this same conscientious class of enlightened practitioners with the most perfect mental equanimity, and it may be, even with somewhat of a spice of self-complacency, quietly sit in their study chairs and unblushingly write out—in true empirical style—prescriptions for mere symptoms in cases of liver disease, just as if they were under the impression that they were prescribing for the case 'rationally' and to the best interests of the patient. How long, I ask, are we to find diseases of the liver even gravely published by otherwise well-educated medical men as 'cases of functional derangement,' as if they really believed that functional derangement was itself a morbid state, instead of being, as it actually is, a mere symptom of a morbid physical condition of some tangible part or another of the hepatic organ—its secreting cells, its ducts, its parenchyma, its blood-vessels, or its nerves? Have medical men, as a class, yet to learn that nothing in nature happens without a cause; that no symptom nor sign ever originates spontaneously; that

every change in function, no matter however trifling it may be, is invariably preceded by a change in the material organisation of some part or other of the tissues inducing it? Although we are not always able to detect it.

I have ventilated these opinions from having long entertained a strong conviction that the slow, snail-like progress of rational medicine along the road to her legitimate goal—namely, that of becoming an exact science—instead of remaining, as she is now, little better than an empirical art, though practised by educated men, is due far more to the errors committed by its teachers than anything else. For if its teachers fail to impress upon the minds of their pupils the advisability of treating the morbid causes themselves, rather than the mere symptoms or chain of symptoms they induce, how can we expect to see rational medicine ever rise sphinx-like from out of the ashes and dust of the past ages of empiricism, from which she has so long, and as yet unsuccessfully, struggled to free herself?

I willingly admit that there was a time, and that too not long since, when medical practitioners, in this and all other countries, had a good excuse for prescribing for mere symptoms; but that time vanished when pathological anatomy proved that mere symptoms were not of themselves morbid states, but merely the result of sometimes widely differing pathological

conditions. Before morbid anatomy opened the window and admitted the light, all diseases were naturally enough christened after their most prominent symptoms. Hæmorrhages from the lungs, stomach, and bladder were respectively named hæmatemesis, hæmoptysis, and hæmaturia, from the causes of these various forms of bleeding being as yet unknown. Consequently their treatment was of necessity, like their diagnosis, empirical. Now, however, the cause of the hæmorrhages being discoverable, and discovered, it is not the act of bleeding, but the cause of the bleeding, which is, and ought to be, prescribed for by the enlightened practitioner; and what now holds good for hæmorrhages I desire to see hold equally good for jaundice, which, like hæmorrhages from the lungs or the stomach, is nothing but a mere sign of a variety of widely differing pathological conditions.

To err is human, and no matter however clever and well trained a man may be in medical diagnosis, mistakes he is sure sometimes to make, and that too more especially in hepatic disease. For not even is the highly-trained 'Scientific Physician' infallible. By this term of 'Scientific Physician,' be it remembered, I do not mean merely members of the promiscuous host of learned and able men who possess in their consulting rooms, besides a stethoscope, thermometer, and microscope, a spirit-lamp, a bottle of nitric acid, and a few test-tubes, but quite another class, and that too,

alas! as yet, a very small one—namely, those physicians who, after having completed the ordinary routine of studies qualifying them to become Members of the Royal College of Physicians, have spent two or three years within the precincts of a practical physiological and chemical laboratory. For to such men, and such men alone, can with any degree of justice be applied the title of 'Scientific Physician.' And I say that even such are by no means infallible in the diagnosis of hepatic affections. I must, however, at the same time do them the justice to add, that as there are a variety of different degrees of fallibility, it may not untruthfully be said that for every time the 'scientific physician' trips and falls, his less educated brother stumbles and falls at least a dozen times. We are still far, very far, from the goal of perfection in scientific medicine. For even in highly educated Germany scientific medicine is but as yet in its infancy; while in England again it may be said to be still in its long clothes, and what is more, if the illiberal Anti-vivisection Act be not abrogated or allowed to become a dead letter, it is likely to remain so for centuries yet to come. The mysteries of the healing art are far beyond the power of even the best collective human skill, unless it be aided by experimental science. For the clue to the unravelling of morbid action can only be found through the study of experimental physiology. Healthy living nature herself

must be interrogated in order to get at the secrets of morbid living action.

Little do the pseudo-sentimental anti-vivisectionists dream of the incalculable mischief they are doing to humanity by decrying the laudable efforts of enlightened medical men to advance the progress of the healing art by studying the functions of the frame on animals. Little do they seem to comprehend that the crusade they are so energetically waging against what they please to call vivisection is not one whit less uncalled for or less unreasonable than the same kind of pseudo-sentimental crusade which was waged against the dissection of the dead human body in the cause of humanity by their equally illiberal and unenlightened predecessors fifty years ago. The present anti-philosophic pseudo-sentimental outcry against vivisection originates in the erroneous idea (partly fostered by a few medical men ignorant of its advantages) that while its cruelties are enormous its benefits are nil. But I, who was a vivisector for ten years, and the first officially appointed teacher of practical physiology in a British medical school,[1] can speak as one having authority on both of these points, and I opine that all the cruelties of the so-called vivisectionists, put together, would not amount in a hundred years to the cruelties perpetrated under the name of sport during one single shooting and hunting

[1] I began the course of Practical Physiology in University College, London, in 1855.

season. While again the advantages of practical physiology to the healing art are simply beyond the powers of human calculation; though not more apparent to the uninitiated eye than the value of an iron nail is to the equestrian. But as Herbert in his 'Jacula Prudentum' says, 'For want of a nail, the shoe is lost; for want of a shoe, the horse is lost; and for want of a horse, the rider is lost'—so say I: For want of vivisection, physiology is lost; for want of physiology, rational medicine is lost; for want of rational medicine, the patient is lost.

Moreover, were it even true (which I emphatically say it is not), as ignorant anti-vivisection sentimentalists assert, that experimental physiology has not as yet enabled its cultivators to solve any of the mysteries of disease, it is assuredly true that it has at least already enabled them to avoid many of the errors that were formerly, and are still daily, committed by those who, ignoring its assistance, trust to their own imaginary transcendental 'practical' acumen. Coupled with the somewhat haphazard sort of assistance derivable from what they please to designate their 'experience.' Which, in the majority of instances, amounts to little better than a routine system, guided by the enlightened 'rule of thumb.' The perusal of the following pages will, I trust, if it does nothing more, at least let one tiny ray of light penetrate through the cloud of their scientific darkness.

In order to be able to do this, it will be necessary for me to begin by making a few remarks on the functions of the liver, the nature of bile, and the physiology of its secretion, so that all my readers may be able to follow with facility the many scientific facts, theories, and arguments which it will be necessary for me to lay before them, in a variety of different forms, and in many different places throughout the work, in support of the new rendering I give to many of the old data, as well as the fresh colouring I give to some of the new.

CHAPTER II.

THE CHEMISTRY, PHYSICS, AND PHYSIOLOGY OF THE LIVER.

From the fact that in every individual case of hepatic disease the liver, at least in some part of the course of the affection, becomes altered in composition, in specific gravity, in weight, and in size, it is absolutely indispensable for the practitioner to know something about these factors in health, as well as to be acquainted with some of their more salient variations in disease. I shall now consider each of them in brief detail.

Chemical Composition of the Liver.

There is no single organ in the human body the chemical composition of whose healthy substance varies so much as that of the liver, and this is readily accounted for when the nature of its functions is properly understood. In order to avoid repetition I shall refer the reader to p. 57, where he will find the hepatic functions, which are four in number, treated of in detail. Meanwhile I shall only remark

that the liver, being an organ of the body intimately connected with the development and nutrition of the tissues, plays a more important part in the animal economy in youth than in age; and hence it gradually diminishes in proportional size and weight as age advances. Moreover, as the important part it plays is in the preparation of the food for assimilation, the contents of its hepatic cells vary from hour to hour, not only according to the state of the digestion, but also according to the quality and quantity of the food taken. Thus, for example, after a fatty meal the hepatic cells are loaded with oil-globules; after a farinaceous meal they contain a superabundance of sugar and amylum; while after a meal of purely animal albuminous food they are cram-full of glucogen, the animal starchy substance formed by the liver itself out of albuminous and other kinds of non-oleaginous foods. This being the case, it can readily be imagined how it is impossible to state with exactitude the chemical composition of a human liver at any fixed period of life, or even at any one particular period of the day. The condition of the organ, however, differs so very much in disease, particularly in cases of fatty and amyloid degeneration, that I shall give the analysis of a healthy human liver to serve as a standard of comparison in cases of disease. The analysis of healthy human liver which I have elected to give as the standard of comparison is the

one which was made by Professor Lionel Beale of the liver of a healthy man who was suddenly killed by an accident. It is as follows :—

100 parts of the fresh liver yielded—

Water	68·58
Solids	31·42

In 100 parts of liver tissue were of—		In 100 parts of dried solids—
Fatty matters	3·82	12·16
Albumen	4·67	14·86
Extractive matters	5·40	17·18
Alkaline salts	1·17	3·72
Earthy salts	0·33	1·05
Vessels &c., insol. in water	16·03	51·01

Dr. Marcet [1] made in another way a comparative analysis of a healthy sheep's liver and a human diseased one. When dried, they yielded the following results :—

	Healthy sheep's liver	Lardaceous fatty human liver
Carbon	44·00 per cent.	62·99 per cent.
Nitrogen	9·12 „	9·00 „
Fat	24·90 „	35·30 „
Carbon in fat	19·00 „	27·18 „

The Specific Gravity of the Human Liver.

The very same factors which influence the chemical composition of the liver likewise influence its specific gravity.

The specific gravity of the liver increases as age advances, from the fact that the hepatic tissues get denser and denser the older the individual becomes. The specific gravity of the organ is, however, liable to

[1] *Path. Soc. Trans.* vol. xxii. p. 12.

great fluctuations in disease at all periods of life, in consequence of its depending in a great measure on the amount of the fat-globules stored up in the hepatic cells. This is most markedly shown by comparing the specific gravity of a piece of normal human liver tissue with a piece taken from a liver in a state of fatty degeneration :—

Normal human liver tissue having a specific gravity of 1·50 = Water 100
Fatty degenerated „ „ „ 1·03 . . . .

There is even found to be a considerable difference in the specific gravity of healthy liver tissues according as they are taken from lean and fat persons, the specific gravity being on an average as 1·3 against 1·5. The effect of fat in changing the specific gravity of the liver tissue will be readily understood when I say that human livers have been known to float on ordinary drinking water, and on analysis have been found to contain as much as 65! per cent. of fatty matter.

Weight of the Human Liver.

Not only does the human liver, like all other organs, vary in its absolute weight at different periods of life, but, strange to say, it also varies in its relative weight to that of the whole body at different periods of life. Like fœtal organs, though in a much less degree, its relative weight diminishes in proportion to the gross weight of the body in a direct ratio as age

advances, and the activity of the vital processes diminishes. This is clearly seen in the subjoined table, which gives the average weights of the human liver and body from birth onwards:—

At birth the liver's weight is as	1	to	18	of	the	whole	body
In infancy	„	„	1	„	20	„	„
At puberty	„	„	1	„	30	„	„
At full growth	„	„	1	„	35	„	„
In middle life	„	„	1	„	40	„	„
In old age	„	„	1	„	50	„	„

Thus showing that the child has, in proportion to its bulk, a liver twice and a half larger than the man tottering in decrepitude to the tomb, which fact of itself proves that some one at least of its functions must be of vastly greater importance to animal life in childhood than in old age. What this function of the liver is, which is so much more important in youth than in old age, no one has hitherto even so much as attempted to guess at. Therefore I may perhaps be pardoned if I venture to suggest that it is the saccharine function, which I shall presently have occasion to show is one of the chief the liver performs in the healthy animal economy. The reason why I fix the blame upon the hepatic saccharine function as being the cause of the liver's diminution in relative size in old age is based upon the well-known fact that it is in early life, while the tissues are developing and all the vital processes are most active, that most sugar is required. Hence it is that children are

so fond of sweets, and that the craving for them steadily diminishes in a greater or lesser degree as age advances and progressing development ceases. However, as I am not going to discuss that point here, I shall now proceed to give a table of liver weights at different ages, which I think will be found to be sufficiently exact to render it of practical service to my younger brethren in the post-mortem room, where I have myself often felt the want of something approaching to a reliable table of comparison. I have drawn the table up from data derived from a variety of sources, and it is as follows:—

Weight of Human Liver in proportion to Age and Weight of Body.

			lbs.	ozs.
Between 1 and 4 weeks of age with a bodily weight of from			7½	it is 6
„ 1 „ 4 months	„	„	14	7
„ 4 „ 8 „	„	„	28	8
„ 8 „ 12 „	„	„	34	10
„ 1 „ 2 years	„	„	40	12
„ 2 „ 4 „	„	„	46	18
„ 4 „ 8 „	„	„	56	30
„ 8 „ 16 „	„	„	68	40
„ 16 „ 30 „	„	„	100	50
„ 30 „ 60 „	„	„	150	52
„ 60 „ 70 „	„	„	120	46
„ 70 „ 80 „	„	„	110	38
„ 80 „ 90 „	„	„	100	34

It may be as well for me here to remark that these weights yield no clue to the weight of the liver in disease ; for while an adult liver may be met with weighing as little as 10 ounces, another may be encountered of the enormous weight of 384 ounces

(24 lbs.!). This last marvellous example occurred in a case of encephaloid cancer in a man aged 50, which Dr. Gordon has reported in the 'Dublin Quarterly Journal' for November 1867. The liver had entirely lost its shape, and looked like a great ball, with encephaloid cancerous nodules projecting from all parts of its surface.

Taking all in all, the average normal weight of the human liver in proportion to the gross weight of the body is said, according to Quain's 'Anatomy,' to be as 1 to 36; and this I consider to be most probably a correct estimate to go by when, from insufficient data, in doubt as to how to make the pathological calculation.

Size of Human Liver.

The actual linear dimensions of a normal liver, freshly removed from the body of a well-developed average-sized man, say a sailor of 5 feet 7 inches, are usually 11 inches in its transverse, $6\frac{1}{2}$ in its antero-posterior diameter across the broadest part of the organ, and 3 inches at its thickest part from above downwards; its absolute bulk is about 90 cubic inches, and its average weight 50 ounces. In a healthy female, again—a servant-girl—of 5 feet 4 inches in height, it usually measures $10\frac{1}{2}$ inches in the transverse, and $6\frac{1}{4}$ in the antero-posterior diameter, and weighs about 45 ounces.

As we cannot, however, get at the liver itself, either to measure, weigh, analyse, or take its specific gravity, while the patient is alive—and this is the very time that the physician wants to ascertain these points—the question is, 'How can we by other means obtain the requisite information?' Fortunately for clinical medicine, the physician has it within his power, under certain circumstances, and with proper precautions, to ascertain with comparative exactitude the intrinsic value of the most important (clinically speaking) factor of all, namely, that of *size*. So I shall now proceed to show how this can be easiest and best accomplished.

In consequence of the liver being a large, well-defined, compact, solid body, surrounded by elastic non-solid organs—lungs, stomach, and intestines—by percussion we can obtain a tolerably exact idea not only of its relative position, but of its actual dimensions. When struck, the tissue of the liver yields a heavy, dull sound, whereas all the surrounding organs—lungs, stomach, and intestines—give out, if not a tympanitic, at least a well-defined resonant tone. Consequently, by a process of comparative percussion, the dull boundaries of the hepatic organ are recognisable in the midst of the resonant area formed by the surrounding non-solid organs, and from a combination of the results of numerous physiological and clinical observations made

upon subjects with healthy livers, it has been found that, by a system of manual percussion, the exact dimensions, not only of a normal but of a diseased liver, can be thus ascertained with approximate exactitude.

It is now a well-determined fact, for example, that the area of hepatic dulness, in healthy well-formed individuals, usually commences at a spot two inches, in a direct line downwards, from the right nipple; and consequently the main calculations of the perpendicular extent of the anterior dull area of the organ are made in this line. Thus, in describing the normal extent of the anterior hepatic dulness in a person of 5 feet 7 inches in height, the dulness is said to be 4 inches; which means the dull area which has been ascertained in the direct perpendicular right nipple line, while the patient was lying in the dorsal recumbent position. Under the same circumstances again, in a person of 5 feet high, $3\frac{1}{2}$ inches are usually put down as the standard of the absolute dulness; while in one of 6 feet or more, $4\frac{1}{2}$ inches are considered to be the full limit of the dull area in the perpendicular right nipple line. In all persons, be they big or little, the left margin of the dull hepatic area usually terminates at a point situated about $1\frac{1}{2}$ inches to the left of the lower margin of the xiphoid cartilage. This measurement is

oftentimes a very uncertain one, in consequence of the presence of the generally distended and tympanitic stomach, the resonant tone from which sometimes completely masks the dull sound elicited from the thin left margin of the liver.

Fortunately for the purposes of the clinical physician, it happens that in the very set of cases where it is most essential that this boundary should be correctly ascertained, the tissues of the liver are as a rule so hardened, and its edge so thickened and rounded, as well as the organ as a whole so enlarged, that its left margin yields such a distinctly dull sound on percussion as to defy its being masked by the resonant sound yielded by the tympanitic stomach.

The same consolatory remark is, however, not applicable to another set of cases, where it is almost of equal importance to ascertain the exact position of the left margin of the liver, namely, in those of hepatic atrophy. Then it is, indeed, that the stomach interferes most in the establishing with exactitude the left lateral dimensions of the liver; but fortunately the atrophying process generally proceeds uniformly throughout the whole organ, so that if one of the measurements of the liver has been exactly ascertained in this disease, all the others can be approximately deduced from it by a simple rule-of-three calculation.

Thus, supposing, for example, that the exact absolute dulness of the atrophied liver in the perpendicular right nipple line has been ascertained to be $2\frac{1}{2}$ inches, the transverse measurement may be approximately ascertained from the relative normal proportions as follows :—

$$4 : 2{\cdot}5 :: 11 : x,$$

and the antero-posterior diameter by

$$4 : 2{\cdot}5 :: 6{\cdot}5 : x.$$

Such calculated measurements, however, can only be applied to the very limited number of cases of disease in which a uniform increase or diminution in the size of the liver is known to occur; they are utterly inapplicable to cases, such as cancer, abscesses, and hydatids, where no uniformity in the extension of the diseased area is maintained. In such cases, from certain portions of the organ being alone implicated, and the remaining portions being left in an absolutely normal state, it is necessary to subject the patient to the fatiguing ordeal of a dorsal, as well as of an axillary, in addition to mere anterior percussion.

I must not omit to here call attention to the fact that the dull area ascertained to exist in the hepatic region, through the medium of simple percussion, does not in all cases represent the exact dimensions of the liver; for the liver may occa-

sionally be perfectly normal in cubic capacity, and yet the dull area in the hepatic region be found either to be in excess, or to be less than what it ought to be, had the dull sound elicited represented the boundaries of the organ. The reason of this is easily explained in the following cases :—

A.—Conditions likely to give rise to erroneous signs of diminution in the bulk of the liver.

The position of a perfectly normal liver in the abdomen of different individuals varies sometimes very considerably, for the following reasons :—

1. The mere difference of sex makes a difference in the position occupied by the liver ; therefore, when arriving at a diagnosis from data obtained from the position and size of the liver in women, it must not be forgotten that the conformation of a woman's chest being different from that of a man's, the normal anatomical position of the liver is different in the abdomen of the woman from what it is in the abdomen of the man, being from one to one and a half inches lower down in the right hypochondriac region, and nearly quite as much below the right nipple, than in a normally formed male subject. I am now speaking from personal observation, and as my experience only extends to civilised women, who distort their chests and abdomens by corsets and straight

waistcoats, the above remark has reference to them alone. It matters to us, however, but little what the position of the liver is in uncorseted savage women, for I should think it very unlikely that many of my readers will be called upon to treat liver diseases among them.

2. Some persons have such unusually lax hepatic suspensory ligaments, that the liver floats about in the abdominal cavity, and has not inaptly received the title of 'movable liver,' analogous to 'movable kidney.' And the possibility of a patient possessing one of these 'movable livers' must not be lost sight of when calculating the extent of the dull hepatic area. For if the suspensory ligament chances to be unusually lax the liver sinks downwards and backwards in the abdominal cavity, when the patient is in the recumbent dorsal position. Occasionally to such an extent as to remove entirely its anterior surface from contact with the abdominal walls, and in consequence thereof a loop of tympanitic intestine may intervene and completely mask the dull hepatic sound, which would otherwise be elicited by percussion. In a case of 'movable liver' of this kind, notwithstanding that the liver is of normal dimensions, it may happen, from only a very limited area of incomplete dulness being perceptible, that the case may be readily mistaken by the inexperienced for one of atrophy. In all doubtful cases of

'movable liver' it is therefore necessary also to percuss the organ, while the patient is in the erect position. For although cases of atrophy are not rare, cases of abnormally loose suspensory ligaments are common in delicately formed individuals—both male and female.

3. The position of the liver in the abdomen varies, even in the same individual, at different times of the day, on account of physiological causes. Thus :—

a. A full stomach presses the liver downwards and backwards from the abdominal parietes.

b. An empty stomach admits of the liver ascending upwards and forwards.

c. Inspiration pushes the liver downwards.

d. With expiration the liver rises in the abdomen.

In the upright posture, the anterior margin of the normal liver sinks from one to one and a half inches lower in the abdomen than the position it usually occupies when the individual under examination is lying flat on his back.

4. Another deceptive semblance of atrophied liver arises from an accumulation of gas, either in the stomach, intestines, or peritoneal cavity. This source of error is best avoided by making a comparative examination of the dull hepatic area while the patient is in a standing position.

B.—Causes likely to lead to the idea of the liver's being enlarged when it is not.

This mistake may arise from :—

1. An accumulation of hardened fæcal matter in the transverse colon, giving rise to a marked continuous dulness on percussion with that arising from the lower margin of the liver. And from the dulness arising from the fæces in the distended colon being added by mistake to the actual hepatic dulness, the liver may be pronounced to be enlarged ; when, so far from being in an enlarged condition, it is in reality actually smaller than it ought to be.

2. The presence of abdominal tumours impinging on the liver is a frequent source of error. More particularly is this the case in fat females with uterine and ovarian tumours of the right side. Even in males, however, serious errors in diagnosis have thus arisen. An enlarged and displaced spleen—the result of malarial disease—as well as a hydronephrosis having been, to my own personal knowledge, mistaken for, and treated as, cases of enlarged liver, by men of no mean diagnostic power in hepatic diseases.

3. As already said, in calculating the dimensions of the liver in females, when it is suspected to be atrophied, the probable effects of tight lacing must

be taken into account. The same remark is equally applicable when hypertrophy of the organ is suspected, for the continuous effect of long stays and tight lacing is to induce a marked and permanent displacement of the liver downwards. While at the same time, from the organ being tilted on its axis, its diaphragmatic surface is brought forwards into contact with the anterior abdominal wall, and thus an impression of hepatic enlargement is often given, where no such enlargement exists. On one occasion, to such a marked extent did this occur in a female of sixty years of age—a patient in University College Hospital—whose autopsy I made, that the physician in charge, though experienced in hepatic diseases, had diagnosed and treated the case as one of chronic 'idiopathic enlargement' of the liver. Which was not in the least to be wondered at, seeing that the lower edge of the right lobe actually reached to the crest of the ilium. At the autopsy a strange state of affairs was discovered. The liver was found to be divided transversely by a deep sulcus, which gave to it, in consequence of the further distortion caused by the lower margin of the tightly laced stays, an hour-glass appearance, as well as communicated to the eye the idea of considerable hepatic enlargement. Whereas when the organ was placed upon the scales it was found to weigh only 40 ounces—that is to say, 5 ounces less than normal.

4. In French workmen, as well as in English sailors, who are in the habit of keeping their trousers up by a belt instead of suspenders, it is no uncommon thing to find the liver with a deep furrow running across it, as in the female just alluded to.

5. Just as such errors as these may arise in calculating the dimensions of the liver downwards and laterally by percussion, so again mistakes may arise regarding the extent and position of the liver in an upward direction, from coexistent disease in the base of the right lung and pleura. The upper margin of hepatic dulness reaches normally to within two inches of the nipple, and in cases of consolidation of the lower lobe of the right lung—from pneumonia, cancerous infiltration, &c.—as well as when an effusion of fluid exists in the pleural cavity, the dulness from these causes is sometimes so markedly continuous with that of the liver as to lead to their being confounded together. Thereby giving rise to the supposition that the liver is enlarged and projecting against the diaphragm, pushing it upwards into the chest, when nothing of the kind has occurred.

6. There is yet another possible source of error, though, from its extreme rarity, an error is unlikely to be committed. But as the mistake is said to have once occurred, and it is quite possible that it may occur again, it is but right that I should call

attention to it—namely, the possibility of mistaking an anteverted small liver for an enlarged one.

Dr. T. D. Griffiths, who related the case where this happened, says that in a man aged 54, 'the liver dulness extended from *two fingers' breadth below the nipple to the crest of the ilium, and partly into the iliac fossa, eight inches in the perpendicular right nipple line.*' Yet at the post-mortem the organ was found to measure only 10 inches transversely, and 8 in the antero-posterior direction, weighing 56 oz. This unusual state of affairs, he says, was due to the 'liver lying on its under surface in the lumbar region. The anterior edge of the right lobe being down in the iliac fossa. The organ appeared as if it had been partly *rotated* upon its own transverse axis ; or, in other words, *anteverted*, so as to allow its upper surface to be felt through the abdominal wall below the ribs.'

7. It may be as well for me here to remark that on examining the livers of patients labouring under heart disease (especially in cases of well-marked tricuspid insufficiency) by means of palpation, it occasionally happens that a distinct feeling of systolic pulsation is communicated to the hand pressed firmly—though gently—on the right lobe of the liver, which pulsation, it is said, has been mistaken for an aneurism. In the case of exposed liver related at page 1036, as there stated I could feel no pulsation

whatever when I applied the palm of my hand to the exposed surface of the patient's liver. Therefore I think that even when a pulsation over the region of the liver is discovered in cases of heart disease, it is much more likely to be communicated to the hand indirectly from the aorta than directly from the hepatic arteries. I think a great deal of needless speculation has been made regarding the pathological nature of the so-called hepatic pulsation.

The following remarks on the subject I chanced to fall upon while reading Dr. Milner Fothergill's work on heart disease (p. 85) in connection with insufficiency of the tricuspid valves. He says, 'All the branches of the venæ cavæ become distended.' The liver becomes engorged with blood, 'and pulsates with the regurgitating current driven backwards by the hypertrophied right ventricle.' Liver-pulsation from venous engorgement leads to enlargement, and ends in the 'tissue becoming firmer and harder than normal. The liver is easily deranged by an excess of food when so affected, and great care in diet is requisite for the proper performance of its functions. There is serous effusion from the gorged venules into the bile-passages attending it, so that Oppolzer has given to this condition the term "albumicholie," and he further states that in this condition there is congestion of the mucous lining of the bile-ducts with jaundice, and that both these conditions are readily

affected by a common cold.' I give this quotation simply as it stands in Dr. Fothergill's book, without making any comment upon it.

8. There is yet another point in connection with hepatic percussion to which I must call special attention, as I am aware from personal experience that it always merits attention; and that is the possibility, nay even the probability, of mistaking the dull sound yielded on percussing contracted recti muscles for that produced by hepatic tissue. The mere fact of calling attention to the probability of such an error will, I think, of itself be sufficient to prevent the possibility of its occurrence.

Lastly, in examining the liver, it ought always to be borne in mind that the co-existence of disease in the neighbouring organs may considerably alter its position, and also that the existence of fluid in the peritoneal cavity greatly interferes with ascertaining its boundaries by percussion.

Armed with a knowledge of these physiological and clinical facts, it is not difficult to ascertain in what respect the dimensions of a diseased differ from those of a healthy liver. By this remark, however, I do not wish it to be supposed that I for one moment mean to assert that an inexperienced hand would be able readily to ascertain, with anything even approaching to exactitude, the correct dimensions of a diseased human liver. For although one accustomed

to percuss the organ can do so with facility, a tyro in general finds it difficult even to map out the exact boundaries of a healthy liver in a well-nourished individual, even where no pain exists to interfere with the percussing process.

The Functions of the Liver, and the Chemistry of its Secretions.

As is well known, the liver is not only the largest secreting gland in the human body, but the one to which has been accorded the prominent distinction of being situated in the very centre of the frame, and placed in intimate connection with both the respiratory and digestive systems. Being directly connected, on the one hand, with the digestive canal by a special venous system of its own, and indirectly connected on the other with the respiratory organs by the general venous system. Directly receiving from the digestive canal certain portions of the food pabulum, which have there undergone the necessary preparatory process of transformation to render them capable of absorption by the numerous portal capillaries—the saccharine and albuminous—and indirectly from the respiratory organs certain other portions of the nutritive elements of the food, which have already undergone in them a still further process of transformation, by having been oxidised in the pulmonary capillaries—the fatty and oleaginous.

The essential importance of the liver to animal life is still further demonstrated by the fact that even while the fœtus is yet within its mother's womb it is to the liver that is directly returned the blood issuing from the maternal placenta. The part the liver plays in the animal economy is in fact proportionately greater than that of any other glandular organ in the body, and this can surprise no one who is acquainted with modern physiology. For it is now a well ascertained fact that the secretion of bile is merely one out of four important functions which the liver has to perform ; to wit :—

1. A sugar manufacturing.
2. A fat modifying.
3. A calorifying, and
4. A bile-forming function.

It is not difficult, therefore, to understand what a great amount of disturbance in the performance of the various bodily functions must arise from the liver ceasing to do its proper work, and how essential it must be for the practising physician to understand the normal hepatic functions aright, if he is expected to be able to treat his patients rationally and on scientific principles. For just as dirt is nothing more or less than good matter out of place, so disease is merely healthy function out of order. Hence, in order to understand diseased action, it is absolutely essential to understand

healthy function. Therefore, before I enter upon the consideration of the liver's disordered action, I shall first speak of each of its normal functions in detail.

The Liver as a Sugar Manufacturer.

It is now well known to every physiologist that no vertebrated animal—fowl, fish, or mammal—can exist without a certain amount of saccharine matter, which saccharine matter, if not supplied to it along with its food, it must possess the power of itself forming out of some one or other of the elements supplied to it in the shape of alimentary materials; and thus it is that while the herbivora obtain a great part of their saccharine supply directly from what they live upon, the carnivora manufacture the saccharine materials they require for their wants out of the non-saccharine alimentary substances they eat. And it is to the liver, the largest, and, as I have already said, the most important, glandular organ in the body, that has been delegated this sugar-manufacturing process. It is indeed the only organ in the body which enjoys the special power of transforming albuminoids into sugars, though others possess the power of transforming amyloids into saccharine matter—to wit, the salivary and pancreatic glands.

Moreover, there are two perfectly distinct kinds of sugar manufactured by the liver: a non-crystal-

lisable, identical with grape-sugar, and a crystallisable, to which chemists have given the name of 'milk-sugar,' from its being normally found in the milk of all animals, be they carnivora or herbivora. Indeed, it is the very fact of the milk of the carnivora containing sugar, which is the best evidence of all that animals possess within their frames a sugar manufactory; for the carnivora certainly cannot obtain sugar from without along with their food.

Although the mammary glands excrete this crystallisable form of sugar, there exists as yet no proof that they manufacture it; on the contrary, indeed, all the evidence yet adduced tends to show that the liver is the only manufacturer in the body of saccharine matter out of albuminoids. Consequently we must accord to it, at least for the present, the credit of manufacturing the crystallisable animal sugar which the mammary glands of the carnivora, as well as of the herbivora, supply to their offspring in the milk.

That healthy human blood, although not always containing sugar, yet always contains glucogen, is easily shown by adding a solution of iodine in iodide of potassium to a drop of blood under the microscope, when the contents of the colourless blood-corpuscles immediately assume a brownish-red tint, from their containing glucogen.

The question of the saccharine function of the

liver is a subject to which I have more or less directed my attention since 1853, when I communicated to the Société de Biologie of Paris an account of an experimental procedure whereby diabetes can be produced artificially in animals by simply stimulating the liver to an excessive production of sugar, by means of ammonia, chloroform, ether, and alcohol, injected into the portal vein. While again, in 1859, I communicated to the Royal Society another series of experiments founded on a different plan, which I regarded as proving the following eight propositions :—

1. Sugar is a normal constituent of the blood of the general circulation.

2. Portal blood of an animal on mixed diet contains sugar.

3. Portal blood of a fasting animal, as well as of an animal fed solely on flesh, is devoid of sugar.

4. The livers of dogs contain sugar, whether their diet is animal or vegetable.

5. Under favourable circumstances, saccharine matter may be found in the liver of an animal after three entire days of rigid fasting.

6. The sugar found in the bodies of animals fed on mixed food is partly derived directly from the food, partly formed by the liver.

7. The livers of animals restricted to flesh diet possess the power of forming glucogen, which glu-

cogen is, at least in part, transformed into sugar in the liver—an inference which does not exclude the probability of glucogen (like starch in the vegetable organism) being transformed into other materials besides sugar.

8. As sugar is found in the liver at the moment of death, its presence cannot be ascribed to a *post-mortem* change, but is to be regarded as the result of a natural physiological condition.

These conclusions have been, since their publication in the 'Proceedings of the Royal Society,' vol. x. p. 289, abundantly borne out by the experiments of other observers; as may be seen, for example, in No. 22, p. 214, of Pflüger's 'Archives,' where the paper of Seegen and F. Kratschmer appears. I may also mention that since the above series of experiments were published, Von Wittich has found that absolutely fresh bile possesses the power of converting boiled starch into sugar. If, for example, from twenty to forty drops of freshly secreted bile be put into a test-tube along with boiled starch, and kept at an ordinary temperature for an hour, when tested with the sulphate of copper solution it gives a distinct saccharine reaction. It is possible, then, that it is the ferment existing in the fresh bile which converts the hepatic glucogen into sugar while it is still within the parenchyma of the liver; and if such in reality be the case, it is

all the more easy for us to understand the *rationale* of the saccharine function of the human liver.

When from any cause the saccharine function of the liver is interrupted, and the transformation of albumen into glucogen, and glucogen into sugar, is interfered with, the hepatic cells get filled with glucogenous albuminoid materials, and the diseased condition of the organ known as 'amyloid' or 'albuminoid liver' is the result. While, on the other hand, when the saccharine function of the liver is abnormally active, and more sugar is formed than the wants of the system demand, the excess is excreted by the kidneys, and the disease which we call 'glucosuria,' or diabetes, is established. The diabetes in this case not being due to a diminished bodily saccharine consumption, but (as described in my book on 'Diabetes') to an excessive saccharine formation. There are two kinds of diabetes, which, in consequence of the differences in their pathology, require of course very different kinds of treatment.

Fat-modifying Hepatic Function.

While the liver not only prepares, but even manufactures some of the saccharine elements of nutrition, it merely modifies those of the fatty and oleaginous groups; and even that it cannot do until they have previously undergone a process of oxidation in the lungs.

The great difference in the power the liver possesses of preparing different kinds of foods for the purposes of assimilation might almost be conjectured from a study of the anatomical arrangements made for their reception by the organ. Thus, as I before said, while the saccharine and albuminous portions of the food are directly taken up by the capillaries of the portal vein, and carried at once to the hepatic cells, the fatty and oleaginous elements of the food are absorbed from the intestines by a special system of vessels called lacteals, which merging together form the thoracic duct, and they are carried by it *past the liver*, and poured into the general venous circulation. At a point from whence they can be at once transported to the pulmonary capillaries and exposed by them in the vesicles of the lungs to the direct oxidising action of the inspired air, before being allowed to come into contact with the hepatic tissues, which are to give the finishing touches to them in their preparation for the purposes of assimilation.

The precise manner in which the hepatic cells act upon the fatty and oleaginous elements of the food is still a mystery. All we know is that the cells, besides under normal conditions preparing them for the purposes of nutrition under abnormal conditions, possess a remarkable power of extracting oil-globules and fat-granules in a free state

from the blood, and not only storing them up in their interior, but transforming them into a beautiful white crystalline substance called cholesterin. Which they excrete along with the bile, sometimes in such quantity, too, that it forms itself into hard concretions—called gall-stones—either in the bile-ducts themselves or in the gall-bladder.

When from some cause or another the fat-modifying function of the liver becomes greatly disordered, the hepatic cells get choke-full of oil globules, and the diseased conditions to which have been given the respective names of 'fatty' and 'lardaceous' livers are the result.

Calorifying Hepatic Function.

Although the rationale of this function would perhaps be more readily understood if I were to delay its consideration until after I had explained the nature of the biliary function—as the introduction of the few necessary words I have to say upon it after I have described the nature of bile and its mode of secretion would break, in a measure, the consecutive chain in the information to be conveyed—I prefer taking the consideration of hepatic calorification up here, on the ground of its being the lesser of two evils.

Everyone knows that no function in the animal

body can be performed without a chemical change of materials. Further, that every chemical change of organic materials is more or less associated with their active oxidation, and yet further, that no form of oxidation whatever can occur without the generation of an amount of heat in direct proportion to the activity and amount of the oxidation. Moreover, from the amount of heat developed being proportionate to the activity and amount of oxidation, it follows, as a natural consequence, that the greater the activity of the chemical changes—which, from their occurring in the animal body, we denominate functions—the higher must be the temperature of the organ in which they occur. Now after having said that the liver is not only the largest, but, as regards the number of its functions, the most important organ in the whole body, it can astonish no one to hear that it is at the same time normally the warmest gland in the human frame. This is due to no peculiar or specific form of vital action going on in it, but solely and merely the direct consequence of the number and variety of the transforming, modifying, and assimilating chemico-physiological processes which occur in the hepatic cells being attended by the absorption of oxygen and the liberation of heat in a free state. The amount of heat so liberated is indeed actually sufficient to raise the temperature of the organ five degrees higher

than that of the rest of the body. I have myself found, in experimenting upon the dog, the mercury in a sharp-pointed thermometer, inserted into the hepatic tissue through a small opening made in the abdominal walls, stand at 105° Fahrenheit; whereas when the end of the instrument was withdrawn from the tissue of the liver, and pushed into the abdominal muscles, the mercury in the space of a few minutes sank down to 100°. It is not to be wondered at, then, that physiologists should regard the human liver as *par excellence* the great calorifying organ of the body.

Having given this brief *résumé* of what some of my readers may perhaps regard as the three minor hepatic functions, I have now to take up the consideration of what they will in like manner, no doubt, consider the major, and consequently to us, as practitioners, the most important, of the hepatic functions, namely—

The Biliary Function of the Liver.

In order that the reader may all the more readily understand the derangements which arise from a stoppage of the secretion of bile, it will be advisable for me to remind him of the chemical and physical properties of a few of the more important substances which are met with in normal human bile.

ON THE NATURE OF BILE.

In a few words, human bile may be said to be composed of the following substances :—

Firstly, biliverdin, a green, nitrogenised, non-crystallisable colouring matter, analogous to the green colouring matter of the leaves and other green parts of plants, and, like it, leaving on incineration—as I was the first to show (1852)[1]—a distinctly ferruginous ash. This bile-colouring matter appears, like urohæmatin and all other animal pigments, to be a direct derivation by simple oxidation of the colouring matter of the blood, and not, as some have erroneously supposed (Frerichs), derived from the transformation of the bile acids into pigmentary matter.[2]

[1] *Vide* paper by the author on the colouring matter of the urine, *Pharm. Journ.* November, 1852. Also my paper entitled

'Urohæmatin, and its Combination with Animal Resin.' *Verh. d. Phys.-Med. Gesellschaft zu Würzburg*, Bd. v. 1854.

[2] In a letter which appeared in the *British Medical Journal* of the 24th April, 1880, Dr. Chas. MacMunn calls attention to the fact that the colouring matter of the urine is partly derived from bile pigment which has undergone the modifying process of oxidation, which fact was already pointed out by me in the above-mentioned papers over a quarter of a century ago.

On this subject I received the following letter from Dr. MacMunn dated

'Wolverhampton, Feb. 28, 1881.

'Dear Sir,

'I beg to call your attention to a paper of mine in the *Proc. Roy. Soc.* No. 208, 1880, which is the continuation of another published in *Proc. Roy. Soc.* No. 206, 1880. I would send you reprints of both, but I know you take in the Proceedings. Thus you will see that your own researches on urohæmatin, published in 1852, are confirmed by means of

Fresh and healthy human bile is usually of a brownish-yellow-green colour, varying, however, in depth of hue, very greatly, according not only to its degree of concentration, but to the state of the system, as well as to the kind of food taken.

As regards the influence of food upon the colour of bile, if I may be allowed to form an opinion from the effects of different foods on the dog's bile I have experimented upon, I should say that, as a rule, animal food tends to give bile a yellow, and vegetable food a more or less distinctly greenish tint.

In some instances even the colour of the food taken is itself communicated to the biliary secretion. Thus it has been noticed that when oxen are fed upon red madder, as they often are in France, the bile taken from the gall-bladders of such of the animals as have freely partaken of the madder has a decidedly red tint. Now this is a point of considerable importance in proving that I am right in thinking that the colouring matter of the bile is not formed by the liver, but is directly obtained from the blood ; for, of course, the red bile can only be derived from the madder pigment through the instrumentality of the

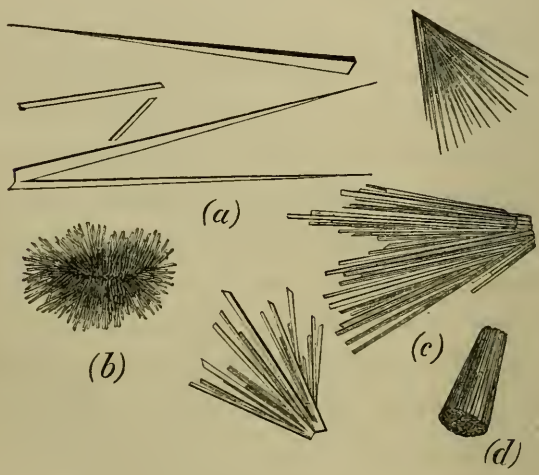
the spectroscope twenty-eight years after their publication. I have not mentioned this in the paper, but all physiologists will at once recognise the fact. . . . So far, I believe it may be taken as established, that all these urinary pigments (with one exception) which are discoverable by means of the spectroscope, can be produced from hæmatin. . . . .

'C. A. MacMunn.'

blood, which in its turn received it directly from the madder in the intestinal canal. This and the following substances are subsequently further spoken of in the chapter on the Chemistry of the Excretions.

Secondly, two peculiar substances, named respectively glycocholic and taurocholic acid—the former yielding, when in combination with soda, a crystal-

Fig. 1.

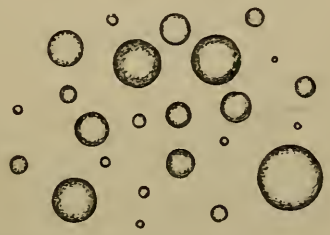


Crystals of Glycocholate of soda, a beautiful polariscopic object. (*a*) Fine needle-shaped crystals, separated from a rosette-shaped group. (*b*) Small rosette of crystals. (*c*) Fan-shaped groups of crystals, which are merely portions of large rosettes that have become broken up. (*d*) A fragment of a bundle of needle-shaped crystals. Mag. 90 diam.

lisable, the latter a non-crystallisable salt. Taurocholic differs still further from glycocholic acid, in containing a large percentage of sulphur, and being, under the influence of hydrochloric acid, convertible

into taurin—a beautiful snow-white crystalline substance. The human body has the further power of converting it into cystin, that beautiful yellowish-green sulphur compound which forms certain kinds of

FIG. 2.



Taurocholate of soda is found in the form of fatty-looking globules of various sizes. They differ from fat and oil globules, however, in being soluble in water, and insoluble in alcohol and ether.

urinary calculi, which are more common in this country than in any other.

Thirdly, cholesterin, a pure white crystalline, fatty matter. Not, however, peculiar to bile, but found in various tissues and secretions of the body—to wit, brain substance, ovarian fluids, &c., and which in abnormal quantity gives rise to gall-stones, as will be fully explained at page 551, where its chemical and physical characteristics will be found given in detail.

Fourthly, a brown resinous substance, resembling, in appearance and consistence, shoemaker's wax, to which I long ago gave the name of 'Hepatic resin' from not knowing what else to call it.

Fifthly, among the constituents of the bile I

must mention sugar, for both in the normal bile of man and of the lower animals, the ox and the dog, I have always detected that substance. On one occasion I even found torulæ in the bile within twenty-four hours after its removal from the gall-bladder of a healthy dog, and the torulæ were of course the product of the fermentation of the sugar contained in the bile.

Sixthly, and lastly, a quantity of inorganic matter, consisting chiefly of soda, potash, and iron.

A. The specific gravity of healthy human bile fluctuates, of course, with the percentage of solid matter it contains. From my own observations I consider that healthy human bile has an average specific gravity of 1020, and contains about six per cent. of solid matter, five per cent. of which is organic, and one per cent. inorganic substance. In diseased states the bile may be of so low a specific gravity as 1012, or so high as not to be calculable by a bilometer from its being as thick as tar or treacle.

B. The reaction of healthy human bile when fresh is almost always neutral, but on standing it rapidly assumes a strong alkaline reaction, in consequence of an alkaline fermentation taking place in it.

THE SECRETION OF BILE.

Firstly, let it be remembered that it is an indisputable aphorism in physiology that every glandular secretion may be either temporarily ac-

celerated, retarded, or totally arrested during the lifetime of the animal. Secondly, that the acceleration, retardation, or arrest may arise either with or without visible change in the glandular tissue. Thirdly, that all forms of glandular secretion are under the direct and immediate influence of nerve force. With these three preliminary aphorisms I shall begin to prove the first proposition, which seems at first sight to be the most difficult of establishment. Namely, the influence of nerve force over the secretion of bile. Yet in reality this is quite as easy a matter as to prove any of the others. (*a*) Is my reader not aware of the influence of fear in arresting his own salivary secretion? Perhaps not. So let me put to him a second question: (*b*) Has he never read or heard of the popular manner of detecting a domestic thief in India? In case he has not, I may as well tell him what every man who has resided in the East Indies knows, namely, that when a household article has mysteriously vanished the servants of the house are all made to stand up in a row, and to each is given a handful of dry rice to chew and swallow, under the popular belief that the guilty man will be unable to swallow the rice. And so he no doubt is, for the simple reason that from the thief's believing in the efficacy of the ordeal, and being conscious of his guilt, he gets into such a state of fright that an immediate arrest of his salivary secretion takes place, so that after rolling

about the rice in his dry mouth, and grinding it with his teeth, from not one single drop of saliva being present, either to soften or to lubricate it, he is at length forced to give up the attempt of gulping the dry grain-dust down his parched throat—and as a natural result his guilt is detected. His honest companions, on the other hand, from their believing that they have nothing to fear, have plenty of saliva in their mouths, and swallow their handful of dry grain with facility. Further, let me remind the reader that there is a not uncommon disease in this country, tolerably well known to physicians, under the name of *ischuria renalis*, in which not a single drop of urine is secreted. Not on account of nervousness, but on account of a morbidly congested state of the parenchyma of the kidneys. Well do I remember in the early part of my consulting career being called to the bedside of an eminent authoress—a patient of Dr. Hastings—who had not passed one single drop of urine for fourteen hours. I immediately catheterised her in order to assure myself that there was actually a total arrest of secretion, and from the bladder I obtained, by means of a flexible male catheter, nothing more than about a drachm of blood-stained mucus. Whereas after applying the requisite treatment for *ischuria renalis*—hot poultices, &c.—no less than forty ounces of urine were passed within the next thirty-six hours, without the employment of any instrument

at all. These, then, are two simple and easily understood examples of arrested, or as it ought rather to be called suspended, secretion, from glandular organs striking work, from two entirely different causes, one nervousness, one structural change. And if such a state of matters can happen to the salivary and renal glands, how can anyone for a moment doubt that a precisely similar thing may happen to the gland which secretes the bile? There is not so much as a shadow of reason to doubt the possibility of the liver's striking work, except a want of physiological and pathological knowledge. Which want, if it at all exists in the reader's mind, I shall now attempt still further to supply.

At page 310, under the heading 'Jaundice the Result of Enervation,' I cite an example of the influence of fear in arresting the biliary secretion in the dog. I there call attention to the fact that if (as Bernard first observed) a dog with a biliary fistula be caressed, the secretion of bile is actively continued; while, on the other hand, if the animal be suddenly ill-used, the secretion of bile is instantly arrested; and if he be again caressed, the secretion is re-established, and the bile flows drop by drop from the end of the canula. Here the influence is entirely produced through the intervention of mental emotion on the nerves of the liver, and through them on the secreting cells of the organ. If such effects as are here de-

scribed occur in the dog, we can surely have little difficulty in understanding how the biliary secretion can be similarly influenced in the highly-developed organisation of the human being. I myself accidentally witnessed a still more remarkable example of the effects of nerve influence in arresting the hepatic secretion, not, as in the above case, from mental emotion alone, but from it combined with physical injury. It happened one day that while I was busy in the physiological laboratory at University College collecting bile from the biliary fistula of a bull-terrier dog, just before going in to lecture, the dog, under an uncontrollable murderous impulse, suddenly sprang upon my favourite tame white rat, which was running about the room—'Eugenie,' an animal of European celebrity, from having been exhibited at several learned societies, both in this country and in France and Germany, as an example of a four-footed beast living without either its supra-renal bodies or spleen, with whom the dog had hitherto lived on terms of peaceful acquaintanceship, if not of actual friendship. On the rat rushing to me and springing upon my knee for protection, I was just in time to save her from the teeth of the dog by the rapid administration of a smart blow on his head with the end of an ebony ruler which I chanced to have in my hand. The dog stopped, staggered, shook his head, and then slunk quietly away into his

box. I thought no more about the matter, but in a short time afterwards, on detaching the india-rubber bag from the biliary canula, I was surprised to find that it contained less than half the amount of bile I had expected, and on looking at the end of the canula, I noticed, to my still further surprise, that not a drop of bile was issuing from the end of the canula. Thinking that its canal had got stopped up, I passed a probe into it, and was still more astonished to find that its course was perfectly free, and that yet no bile flowed. After waiting a time, the lecture-bell rang, and I had to proceed to lecture with what little bile I had procured, leaving the dog behind in the private room along with Eugenie, who was now securely put back into her cage so as to be safe from his unpleasant attentions. The lecture finished, before preparing to go home I again turned my attention to the dog, who from bearing no malice had greeted my return with a friendly wag of his tail, and to my surprise I found that the secretion was now going on as rapidly as before I had administered the blow to him. Here then is an admirable example of how physical injury to the brain—most probably coupled with mental emotion—through the instrumentality of the pneumogastric nerve has the power for a time of totally arresting the biliary secretion. These then are physiological results explanatory of how the liver may strike work and give rise to a suppression of the

biliary function. But as in neither of these instances was jaundice a result of the suppression, I shall now turn my attention to pathology, and call it in to my aid in demonstrating conclusively that there does actually exist such a thing as jaundice from suppression of the biliary function. In order to do this satisfactorily I require to prove that where no bile whatever is secreted by the liver, the skin of the patient becomes of as intensely yellow a tint as it does when the whole system is loaded and engorged with an excessive secretion, associated with an arrested elimination of the bile by the natural channels. In fact I must be able to give positive evidence of the actual existence of well-marked jaundice occurring in a case where there is undoubted proof that no bile whatever is secreted. This is clearly and conclusively shown at page 104. To which I beg to refer the reader, in order to save taking up unnecessary space by repetition.

C. The quantity of bile daily secreted by a healthy human adult has been variously stated in text-books, some giving the daily average amount as low as twenty, others as high as fifty ounces. Judging from the results of my experiments made upon dogs fed on a mixed diet in order to bring them as near as possible under the same dietetic influences as human beings, I think that the average amount may be said to be about one-fiftieth of the gross weight of the body. So that a

person of 100 lbs. weight may be supposed to secrete two pounds of bile daily, one of 150 lbs. weight three pounds, and so on. And I think, in absence of any crucial experimental evidence on the human biliary secretion, we may be content to regard these figures as being sufficiently correct for all clinical purposes.

D. Next, as regards the manner in which bile is secreted. For a long time it was thought, and, indeed, some people think still, that bile exists pre-formed in the blood, and that the liver only excretes it, as the kidneys excrete the urinary ingredients. Another class, running to the opposite extreme, believe that the liver is not merely the excretive, but also the formative organ of the bile. It appears to me, however, that neither of these extreme views is correct, and that the truth lies between the two.

It is, in fact, not at all difficult to prove that the liver manufactures certain biliary constituents, and that it merely excretes others from the blood in which they exist pre-formed. Thus, for example, the two substances glycocholic and taurocholic acids are never to be found in quantity either in the blood, tissues, or fluids of the healthy organism, with the single exception of those of the liver and gall-bladder; and after extirpation of the liver from an animal neither of these acids is to be found in its body at all. Such a substance as cholesterin, on the other hand, which

is not peculiar to the liver or its secretion, but is the product of several organs of the body, is always to be detected in the blood, independently of the presence or absence of the liver. While biliverdin again, the colouring matter of the bile, exists in normal blood, and appears to be nothing more or less than a lower oxidation stage of the ferruginous albuminoid which constitutes hæmatin, the normal colouring matter of the blood. That bile pigment exists pre-formed in the blood does not admit of a shadow of doubt; for, as will be subsequently shown, p. 105, when the biliary secretion is arrested in the human subject, not only does the serum of the blood become deeply tinged yellow on account of the accumulation in it of the bile pigment, but the abdominal serum, the urine, and even, it may be, the milk, all become of a yellow hue, as in a patient suffering from ordinary obstructive jaundice. All these facts when taken together clearly prove that the liver is a formative as well as an excretive organ to some, and merely an excretive organ to others, of the normal biliary constituents.

Lastly, the general opinion is that the secretion intermits, and, like the gastric and pancreatic juices, bile is only formed during digestion. Were it so, however, where would be the necessity for a gall-bladder? Is it not to store up the secretion formed in the intervals of digestion, and to retain it until it

is required? No doubt there are several animals, such as the horse, the deer, the elephant, the rhinoceros, the dromedary, and the camel, as well as many birds, such as the ostrich, the parrot, and others, which possess no gall-bladders at all. On the other hand, again, some animals, even of the same species, by a freak of nature possess two gall-bladders, while less fortunate members of the same family have to do without any at all. Dr. Crisp, in a paper 'On the Morbid Conditions of the Bile and Gall-Bladder,' published in 1850 in the 'Medical Examiner,' tells us the curious fact that while two of the giraffes that died in the Zoological Gardens were found to have no gall-bladders, a third one, on being examined like his fellows after death, was found to be in possession of two!

In animals which have normally no biliary reservoir there is a special arrangement of the digestive apparatus, which renders the presence of a gall-bladder unnecessary. In fact, it is easily shown that the biliary secretion in ordinary cases is continuous; for if in an animal possessing a gall-bladder a biliary fistula be established, and the secretion of bile carefully watched, it will be found that at no period of the day does it entirely intermit, although it is more active at one time than at another. The minimum of its activity being during sleep—the maximum during

active digestion. The absolute quantity of bile secreted in the twenty-four hours is tolerably uniform, being on an average about 50 ounces in a human adult, although the daily amount is slightly influenced by the kind of food.[1]

The Quantitative Constitution of Bile.

The relative proportions of solids to liquids, as well as the relative proportions of the solid constituents to one another, vary considerably, not only in different individuals, but in the same individual at different periods of life, on different kinds of food, and at different stages of the digestion, so that no absolutely fixed standard of quantitative constitution of bile can be given. However, an approximative standard can be arrived at, and the two subjoined analyses are those I have elected to go by, as they are not alone the work of different individuals, but while they give the constitution of the bile in healthy persons at almost identically the same period of life, they give it in opposite sexes and of different nationalities.

The first analysis was made by myself of the bile taken from the gall-bladder of an exceedingly handsome and well-nourished servant-girl, aged 20, a resident in London who committed suicide in consequence of finding herself pregnant. The second was made

[1] Arnold found that dogs secreted more bile on a bread than on an animal diet. (*Zur Physiologie der Galle.* Mannheim, 1854.)

by Frerichs of bile from the gall-bladder of a German, aged 22, who was killed by an accident.

Composition of Healthy Human Bile of an Englishwoman aged 20.
(George Harley.)

Water			920·2
Solids:			
Pigment, Mucus, Glycocholic acid, Taurocholic acid, Biliary fats	Organic substances	60·3	70·8
Soda, Potash, Lime, Iron	Inorganic substances	10·5	
			1000·00

Composition of Healthy Human Bile of a German aged 22.
(Frerichs.)

Water			859·2
Solids:			
Glycocholate and Taurocholate of soda		91·4	140·8
Bile pigment and mucus		29·8	
Fat		9·3	
Salts		7·7	
Cholesterin		2·6	
			1000·00

N.B.—Compare these analyses with that of old people's bile given at pages 791–2.

Several physiologists have given it as their opinion that bile is not essential to life, from the fact that animals have lived for many months after the artificial establishment of a biliary fistula, through which the bile was allowed to flow away and be entirely lost to the system. Now, although this latter fact

is perfectly true, yet it is at the same time evident that the uses of the bile cannot altogether be dispensed with, for animals with a biliary fistula invariably lose flesh, become emaciated, and weak. The hair falls off, the bowels become irregular, while at the same time a great and an almost constant discharge of foul-smelling gases takes place from the intestinal canal. At length, after a shorter or longer period, the animal sinks and dies. The fatal termination can, however, be for a certain time retarded by allowing him an additional quantity of nourishing food. For death from want of bile, as is too often seen in the human subject, is nothing else than death from slow starvation, inasmuch as the food is not properly prepared for assimilation without the assistance of bile.

The fact just related regarding the beneficial effects of an additional quantity of food in prolonging life should never be lost sight of in the treatment of cases of obstruction of the gall-ducts, for, by attending to this circumstance, it is often in the power of the medical man to keep his patient alive for a considerable time longer than would otherwise be the case.

It may perhaps not be out of place if I here briefly enumerate the chief uses of bile in the animal economy.

In order to live, not only must the individual particles of our frames die, but they must be con-

tinually replaced by new materials of a similar kind ; and for the accomplishment of this important end, nature has endowed animals with a digestive apparatus in which their food undergoes the various physical and chemical changes necessary to its absorption and assimilation. In the animal laboratory, or digestive apparatus, there are five important agents constantly at work—saliva, gastric juice, bile, pancreatic fluid, and intestinal secretion, and each of these agents has a special and definite office to perform in the elaboration of the food.[1] At present, however, I limit myself entirely to the consideration of the bile.

Bile is the first digestive agent with which the food in the shape of chyme comes in contact after leaving the stomach and entering the intestines, and immediately on the chyme, which is acid, mixing with the alkaline bile, a white flocculent emulsion is formed, which emulsion has been described by many writers as a precipitation of the albuminose (digested albumen). Careful researches by myself and others have, however, shown that it is not the bile which precipitates the albuminose, but the acid of the chyme, which in reality sets free and precipitates certain ingredients of the alkaline bile. In the majority of cases this is

[1] For an explanation of these offices, see the author's article on 'The Chemistry of Digestion,' in the *British and Foreign Quarterly Review*, January, 1860.

not, however, a true precipitation. For on throwing the milky-looking turbid mixture upon a filter, I found that in the majority of instances almost nothing remained behind, and the filtrate was nearly as white as the original liquid. Further, if the albuminose be separated from the chyme, and the chyme then brought into contact with the bile, the same flocculent-looking milkiness still appears. Nay, more, on adding equal parts of sheep's bile (fresh) to gastric juice drawn from a dog's stomach in full digestion, the apparent flocculent precipitate still appeared, although the acidity of the gastric juice remained unneutralised; and on throwing the whole on to a filter, I found that the liquid that drained through was as milky and flocculent-looking as the original.

Although bile can convert starch into sugar, its chief office in the digestive process is not so much to act on the amylaceous or albuminous portions of our food, as to assist in the absorption of fats.

While speaking of the properties of the bile, I may mention that, although bile has no digestive power (properly speaking) over albuminous substances, yet, when injected into the subcutaneous cellular tissue of a healthy animal, I found that it eats its way out through the skin, just as gastric juice or lactic acid does under similar circumstances. Even the muscles with which it comes into contact appear to be eaten, that is to say, digested away.

When fresh bile is mixed with neutral fat, little change is observed, but when brought in contact with the fatty acids, an immediate emulsion takes place. Lenz and Marcet[1] pointed out how the neutral fats of our food are transformed into fatty acids during their sojourn in the stomach; and Bidder and Schmidt[2] illustrated by experiments on dogs the important part played by the bile in their absorption. A dog, which in its normal condition absorbed on an average seven grains of fat for every two pounds of its weight, absorbed only three grains, or even as little as one grain, after the bile was prevented entering the intestines, in consequence of a ligature being applied to the gall-duct.

Further, these last-named observers found that, while the chyle in the thoracic duct of a healthy dog contains thirty-two parts of fat per thousand, that in the thoracic duct of a dog with a ligatured gall-duct contains only two parts per thousand. These facts clearly prove that bile plays an important part in the absorption of the fatty portion of our food. Next comes the question, 'In what manner does bile aid in the absorption of fatty matter?' As everyone knows, fats and oils have no tendency to mix with water, and hence diosmose between an aqueous and an oily fluid through a membrane is next to impossible. Matteucci

[1] *Vide* a 'Discourse on the Chemistry of Digestion,' by Dr. Marcet, *Journ. of the Chem. Soc.*, Oct. 1862.

[2] *Die Verdauungssäfte und der Stoffwechsel.* Leipzig, 1852.

has, however, shown that if an animal membrane be rubbed over on both sides with a weak solution of potash, it allows oil to pass through it. It has also been observed, that when the walls of the intestine are moistened with bile, they allow oil to pass through them, which would not otherwise be the case. To study this property of bile, I performed the following experiments :—

Firstly,—A clean piece of duodenum was filled with oil, ligatured at both ends, and suspended in water, holding in solution a small quantity of albumen. (The albumen was added to the water merely to imitate slightly the albuminous blood.) On examination, twenty-four hours later, no oil was found to have escaped through the intestinal walls.

Secondly,—Another portion of intestine had its internal surface moistened with sheep's bile before the introduction of the oil. It was then treated in the same manner as the preceding, and on being examined after the lapse of twenty-four hours, a small quantity of the oil was found to have penetrated through the intestine.

Thirdly,—Into a third portion of intestine were poured equal parts of sheep's bile and chyme—obtained from a dog in full digestion through a fistulous opening into its stomach—after being treated for the same length of time and in precisely the same manner as the others, evident signs

of the oily matters of the chyme having passed through the walls of the intestine were obtained, for the fatty matters were seen as a scum floating on the surface of the albuminous water. Moreover, the fatty matters were in the form not of pure oil, but of a soapy substance.

The bile was thus seen to possess one of the more remarkable properties of the pancreatic juice, namely, the emulsioning of the fats of the food. There is this important difference between the action of these two secretions on fats, however, that while bile merely emulsions and saponifies that portion of our food which enters the duodenum in the form of fatty acids, pancreatic juice, on the other hand, possesses the power of emulsioning and saponifying not only the fatty acids, but also the neutral fats; indeed, its power seems chiefly to be exerted upon the latter. Hence it appears that both secretions are in a measure necessary to the complete digestion and absorption of the oleaginous constituents of our food.

A great deal of unnecessary importance has been attached to the fact that a portion of the bile thrown into the intestines during the process of digestion is reabsorbed into the blood along with the food, to be ultimately eliminated by the kidneys as effete matter. Why writers attach so much importance to this fact appears to me a mystery—except it be accounted for

by the ungenerous supposition that they are ignorant of the equally patent fact that portions of every one of the digestive secretions, saliva, gastric juice, pancreatic juice, and intestinal secretions, are in like manner reabsorbed into the blood along with the food, to be eliminated from it as effete matter by the kidneys. I merely call attention to this circumstance in passing, lest some of my readers might be surprised if I omitted to allude to it altogether, which I certainly might have done with little disadvantage to them, as the quantity of bile reabsorbed from the intestines is a mere bagatelle. From the fact that by far the greatest portion of the bile thrown into the intestines during the digestive process is simply voided as useless material along with the fæces. In proof, however, that a certain amount of bile is taken up by the intestinal absorbents and carried along with the food into the general circulation, I may mention that MacMunn has detected, by means of the spectroscope, the effete bile pigment —urobiliverdin—in blood serum, in the same way as he found urohæmatin—the effete pigment of the blood itself.

On one occasion, while experimenting with bile in my laboratory at University College, I was surprised to hear Minton, the servant who was assisting me, say that while he was travelling with Sir Andrew Smith in South Africa, he had oftentimes seen

the Caffres drink bile direct from the gall-bladders of the animals killed by the European party, and that, while passing the gall-bladder round like a loving cup to each other, they would rub their stomachs and say—'Mooé-ka-kolla,' signifying thereby that it was very good. Since then, I have read, at page 155 of Petherick's 'Egypt, the Soudan, and Central Africa,' that the Arabs and natives of Hasanyeh, in the Soudan, use bile as a condiment to their food. He says that he has seen them, after killing a sheep and ripping it open, take out the stomach, liver, and lights—after only shaking out the contents of the stomach—cut them into small pieces, and put them into a wooden bowl, then take the gall-bladder, as a substitute for lemon, I suppose, and squeeze out its contents over them, add a copious supply of red pepper, and immediately proceed—while they were yet warm—to eat them; and that too, apparently, with great relish.

It certainly seems at first sight very extraordinary that a human being should not only drink, but drink with pleasure, a liquid so bitter and nauseating as bile. Perhaps these poor Africans, however, enjoy the sickening-tasted bile for the same reasons as the cattle in their country, at certain periods of the year, go thousands of miles to drink at the brackish salt-springs. There being scarcely any chloride of sodium in the earth in these countries,

there is insufficient for the animal requirements in the herbage on which they feed, and they are forced thus to supply the deficiency by artificial means. Bile contains a large percentage of soda, and perhaps these two tribes of Africans drink it in order to obtain that substance, just as the cattle drink the brackish water of the salt licks, feeling that it agrees with them, without knowing why. We know that in some parts of Africa the natives will barter a handful of gold-dust for an equal amount of common salt, and this can be for no other reason than that they have a craving for it ; while this fact also well accounts for the social importance attached by Arabs to the fact of a stranger having partaken of a man's salt ; for as here shown, instead of the giving a man salt being a mere bagatelle, as it would be in this country, it is there tantamount to giving him one of the most valuable things you possess.

One of my patients—Mr. J. De Villiers—who has lived all his life, until the year 1880, in South Africa, tells me that he has seen horses in the Orange Free State actually eat their own and each other's harness, made of native leather—which is tanned by repeated saltings—for no other reason than to obtain the salt from it. This fact, therefore, entirely confirms my views of the reason why the Africans drink and relish bitter gall. He also tells me that the native idea in Caffreland regarding gall

drinking is that by drinking the bitter gall they protect themselves from the deadly effects of snake-bite and poisoned arrows. Which, of course, is a mere chimerical idea, originating in their ignorance of the uses and actions of bile in the animal economy; but nevertheless one which it may be useful for parents to encourage, in order to induce their children to drink of the nauseating stuff, knowing from experience that it will do them good.

If the preceding chemical, physical, and physiological data are kept clearly in view, I think we are prepared to enter profitably upon the scientific consideration of the mechanism, diagnosis, and treatment of diseases of the liver; and I shall commence with the consideration of the—

CHAPTER III.

ETIOLOGY OF JAUNDICE.

Before entering upon the consideration of the etiology of jaundice, in case some of my readers may be surprised at my giving such prominence to a word which after all is the name of a mere symptom, especially after having said so much against the pernicious system of raising mere symptoms to the dignity of diseases, I think it necessary for me to repeat what I said in my original monograph on this subject—namely, although I here make use of the word 'jaundice' as if I regarded the pathological condition it indicates as being an actual disease, it is only in its mode of employment and nothing more. For, on the contrary, I look upon jaundice in precisely the same light as I regard the terms 'oxaluria' and 'albuminuria,' which are not of themselves diseases, but only the most prominent and the most characteristic symptoms of several widely differing pathological conditions. In like manner the peculiar state of body characterised by yellow skin, saffron-coloured urine, and pipeclay stools, I regard not as itself a disease, but as a mere

symptom of several widely differing morbid states. It may be asked, 'Then why do you speak of jaundice as if it were a disease at all?' To this I reply, 'Because it is commonly spoken of in books as such, and because, although the condition of the skin called jaundice be merely a manifestation of morbid action, and one too requiring neither skill nor experience to detect, the proper comprehension of its true mechanism is of great practical importance to the physician. For without this knowledge it is impossible for him to treat with the slightest chance of success any of the severer forms of hepatic disease giving rise to it. Nay, even his remedies for the symptom jaundice may become dangerous weapons, if unskilfully applied. In fact, it is almost unnecessary to apologise for speaking of jaundice as if it were a disease *per se*; for, notwithstanding all that has been written upon the subject, it is universally admitted that hitherto the simplicity of its diagnosis has only been equalled by the obscurity of its pathology and the uncertainty of its treatment; and no one at all conversant with the literature of jaundice can be in the least degree surprised at this statement. On the contrary, on glancing at the immense variety of morbid states and known pathological conditions with which it is associated, he cannot fail to perceive its cause as well as to admit its truth.

Some of the pathological conditions are closely

allied; others are widely separated—so widely, indeed, that at first sight it is impossible to discover from whence emanates the common symptom.

We find jaundice connected with diseases of the liver, of the neighbouring organs, and of the general system—even such as fevers and gout. In some diseased conditions, jaundice presents itself when it is least expected. At other times it is absent when, apparently, it ought to be present. On the other hand, again, while there are cases in which jaundice is evidently merely a symptom, there are others in which it seems to be almost in itself the disease. We have temporary jaundice from transient derangements. We have permanent jaundice from stationary causes. There are cases in which the cause of jaundice is visible after death to the naked eye. There are others where the minutest search is baffled in ascertaining the cause.

That this is no exaggerated view of the case the following table will show :—

JAUNDICE IS MET WITH,

Firstly, *In Diseases affecting the Liver*—

(*a*) Cancer.
(*b*) Tubercle.
(*c*) Cirrhosis.
(*d*) Inflammation.

(*e*) Atrophy.
(*f*) Amyloid, and
(*g*) Fatty degeneration.

Secondly, *In Diseases of the Bile Ducts*—

(*a*) Congenital deficiency.
(*b*) Accidental obstruction. Arising from gall-stones, hydatids, and from foreign bodies (such as cherry-stones and entozoa) entering from the intestines.
(*c*) Cicatrised ulcer of the duodenum, occluding the orifice of the bile-duct.
(*d*) Tumours of the pancreas, interrupting the passage of the bile.

Thirdly, *In Affections of other Organs of the Body exerting an Influence on the Biliary Secretion*—

(*a*) Diseases of the nervous system.
(*b*) Diseases of the lungs.
(*c*) Diseases of the heart.
(*d*) Imperfect establishment of the extra-uterine circulation.
(*e*) Dyspepsia.
(*f*) Torpidity of the bowels, and consequent accumulation of fæces in transverse colon.
(*g*) Pregnancy.
(*h*) Ovarian tumours.

Fourthly, *In a variety of Zymotic Diseases*—

(*a*) Epidemic jaundice.
(*b*) Contagious jaundice (Yellow fevers).

(*c*) Typhus.

(*d*) Pyæmia.

(*e*) Ague, &c.

Fifthly, *As a Result of the Injurious Effects of certain Poisons*—

(*a*) Snake-bite and fish poisons.

(*b*) Phosphorus.

(*c*) Copper.

(*d*) Lead.

(*e*) Antimony.

(*f*) Ether.

(*g*) Alcohol.

(*h*) Chloroform, &c.

Can it be wondered, then, that a state so easily diagnosed is nevertheless so difficult to comprehend?

Notwithstanding the apparent incongruity of the diseases with which the one common symptom of jaundice is associated, I trust in the following pages to be able to reconcile these discrepancies, and prove that the seeming pathological discord is but 'harmony not understood.'

All physicians, I think, admit that the peculiar state of the skin to which the name of jaundice has been applied, is essentially due to some derangement of the biliary function, the exact nature of the derangement being alone the point of contention. I need not, therefore, waste the time of my readers—who, I presume, are already qualified practitioners—

by giving an account of the literature of jaundice. Even while discussing its pathology, I shall strictly limit myself to the consideration of the opinions at present held by the more advanced of our modern pathologists. The object of this treatise being merely to give a brief *exposé* of my own views, and to point out how modern physiology and chemistry have not only thrown a new light on the pathology of jaundice as well as all the other morbid states connected with liver diseases, but have also given a clue to their more successful treatment.

Frerichs, Murchison, and Legg, in their elaborate treatises on diseases of the liver,[1] say that jaundice may result from one of the three following conditions :—

Firstly,—Obstruction to the escape of bile.

Secondly,—Diminished circulation of blood in the liver, and consequent abnormal diffusion. Each of these conditions giving rise to an increased imbibition of bile into the blood, and in both cases the liver being more or less directly implicated.

Thirdly,—Obstructed metamorphosis, or a diminished consumption of bile in the blood.

From this it is seen, that the pathology of jaundice, according to Frerichs, Murchison, and Legg,

[1] Frerichs' *Clinical Treatises on Diseases of the Liver*. New Sydenham Society's Translation, vol. i. p. 93. Murchison, *Lectures on Diseases of the Liver*, 2nd ed., London, 1877. Wickham Legg, *On Bile, Jaundice, and Bilious Diseases*, London, 1880.

who strictly adhere to this view, is very different from what was formerly taught. For while they have entirely laid aside the theory of jaundice as a result of suppressed secretion, they have introduced two perfectly new elements—namely, abnormal diffusion and diminished consumption. The latter theory being, of course, founded on the supposition that bile, after playing its part in the digestive process, is reabsorbed into the circulation, again to perform some other function in the animal economy, before its final excretion from the organism as effete matter.

The theory of jaundice hitherto most favoured in England, which found such an able exponent in Dr. Budd, and which I have persistently, and not without good cause, I think, advocated for the last twenty years, both by pen and tongue, is, that the disease may arise in two ways—firstly, by a mechanical obstruction to the passage of bile into the intestines, and the consequent reabsorption of the retained bile into the blood ; and secondly, by a suppression of the biliary secretion arising from some morbid condition of the liver itself, whereby the biliary ingredients, from not being eliminated, accumulate in the circulation and stain the skin. Now, although I am not prepared to admit the justice of the views held regarding the origin and function of bile, on which these opinions were originally based by Dr. Budd, I never-

theless believe that in the following pages I shall be able, by the aid of modern medical science, to prove the correctness of Dr. Budd's conclusions themselves, as well as prove that Frerichs's theory is quite incompatible with the new facts that have been gleaned regarding the etiology of jaundice from modern research in the realms of morbid anatomy, as well as in those of experimental physiology.

Seeing that three such able practical men as Frerichs, Murchison, and Legg, completely failed to grasp the value of the new data upon which the idea of retaining as correct the old division of jaundice from obstruction and jaundice from suppression is founded—and which I had, it now seems, vainly flattered myself I had twenty years ago made perfectly plain in my monograph on jaundice—no one can be in the least degree surprised that many recent writers on, and reporters of, hepatic cases (among ordinarily educated practitioners) should appear to be not only hopelessly in the dark as to the true nature and signification of the two divisions of jaundice, but even somewhat confused regarding the value of the facts upon which the theory of the two pathological conditions is based.

I shall now, therefore, once more endeavour to make the thing intelligible, and perhaps this time I may be successful, and that all the more easily too from my having made the discovery that the weak points in my former argumentation did not actually

lie in what I said, but in what I left unsaid, unfortunately, as it now appears, under the mistaken notion that my readers were perfectly familiar with the physiology and rationale of normal glandular secretions, which the subsequent writings of several otherwise able authors on hepatic cases have proved to be an error.

For example, in the chapter on jaundice from suppression, at page 244, I find that Dr. Legg, one of my old pupils, is a disbeliever in the doctrine, and actually goes so far as to say that 'the notion of a jaundice from suppression of secretion fell like that of the old and effete theory that bile and all other secretions were formed in the blood.' I think, however, that after he reads and carefully reflects upon the data presently to be adduced, he will find good cause to omit the above passage from the next edition of his work, in which he otherwise displays a deep acquaintance with hepatic literature.

What it now therefore behoves me to do is: firstly, to show that the biliary secretion can actually be retarded, and even totally arrested, without alteration of hepatic tissue. Secondly, to prove that when the liver strikes work, and secretes no bile, the animal body becomes jaundiced, as a direct consequence thereof. To do this satisfactorily would at first sight appear to be exceedingly difficult if not even quite impossible. And so, no doubt, it would be had I mere pathological data alone to depend upon; but

with the aid of modern experimental physiology the matter is not only brought within the range of philosophic possibility, but even made comparatively easy of absolute proof. In order to clear up the point satisfactorily I must this time begin by supplying the deficiencies in my former argumentation; and I think that by so doing and at the same time not only marshalling my physiological and pathological data in strict logical order, but putting them forward in a graphic and concise manner, it will be next to impossible for any one in the possession of an average amount of physiological and pathological knowledge either to misunderstand them or fail to grasp their clinical as well as their scientific import. This can be done by adducing a case of jaundice where after death the gall-bladder and bile-ducts have not only been found empty of bile but filled with their own white mucous secretion, and illustrating its pathology by placing it side by side with one of jaundice from obstruction, where after death the gall-bladder and the bile-ducts have all been found filled with pure bile.

Now, in order that my facts should, like Cæsar's wife, be beyond the reach of suspicion, I shall require to bring forward a case of jaundice with the biliary passages filled with white mucus and another filled with black bile from among cases published by entirely independent observers. I have therefore carefully gone over the literature of the subject, and,

most fortunately for the interests of scientific truth, from the midst of our own modern English literature I can adduce a most typical example of a well-marked case of jaundice from suppression, complicated with stricture of the bile-ducts, which has quite recently been reported by a most competent observer. Most luckily, too, it is a case which shows in an extreme degree the effects of an occluded duct, associated with a total arrest of the biliary secretion. The case I allude to was published in the 24th volume of the 'Pathological Society's Transactions' by Dr. Moxon, and I shall now proceed to quote from it all the salient facts necessary to convince even the most sceptical reader, if he be capable of conviction at all, that the theory of jaundice the result of suppression must henceforth and for ever cease to be regarded either as a clinical or a pathological myth of the imagination, by any one at least having the slightest pretence to the possession of a clear judgment, coupled with an average amount of medical knowledge.

Dr. Moxon's case is entitled 'Simple Stricture of the Hepatic Duct, causing Chronic Jaundice and Xanthelasma,' and therefore cannot, from its title at least, be suspected of having been published with the view of taking a part in the discussion in which I am now engaged, or with the object of furnishing a weapon either for attack or for defence in the cause I am about to employ it. Had its reporter intended

that it should be so employed, I think that he would most probably have given to it the title of 'A Case of Jaundice from a Total Suppression of the Biliary Function, accidentally complicated with a Stricture of the Hepatic Duct.' Which title would not only have been equally appropriate, when the case is viewed from another side, but equally true, as will immediately appear from the extracts I shall make from it.

The patient, a man of 32 years of age, when admitted into Guy's Hospital, was of a dark, dusky-looking, deeply-jaundiced colour, although he stated that in his youth he was both fair and freckled. He had been at sea for eighteen years, and fourteen years ago had been laid up, while stationed in the Mediterranean, with a severe attack of fever, lasting for three months. He never had syphilis, but he used to drink freely—sometimes seven or eight glasses of 'brandy and soda' in the day. His present illness commenced while at Portsmouth sixteen months before his admission. He then lost his appetite and felt sick. After this condition had lasted about eight months he was attacked with colic in the pit of his stomach, and immediately afterwards he began to be jaundiced. Three months later he had a second and much worse attack of colic, and although he never again had a return of the colic he often had pain in the epigastrium. He was much troubled with itching of the skin when he came to Guy's, which was a month before his

death. Though suffering but little pain, he had all the other symptoms of obstructive jaundice, and in addition 'xanthelasma was plentifully developed in hands, scrotum, and back.' Two weeks before he died he had frequent hæmorrhages from nose, bowels, and bladder, which gradually increased in severity, and he died from exhaustion, attended finally with coma. At the post-mortem the liver, though of normal shape, was large and finely lobulated, as if from cirrhosis, and on its surface were 'numerous large dilated tubes full of clear, watery, colourless, slightly mucoid fluid. . . . One ran up in the peritoneum round the gall-bladder; but the gall-bladder itself was flat and empty, having only a little quite colourless mucus in it. The gall-ducts throughout the organ were excessively wide, so that on section of the liver their contents welled up in enormous quantity. It being a white clear fluid was a strong contrast to the serum of the blood, which was golden yellow. This contrast between the contents of the bile-duct and serum of the blood was most remarkable. The gall-ducts had xanthelasmic-looking patches within them —that is to say white opaque patches. The hepatic duct at the point of union of its two divisions was swollen from the presence in it of a firm tough matter, making a little soft knot of the size of an almond. The duct was here bent, and a fine probe would only pass with care and some force. The thickening was

entirely limited to the duct. The adjacent vein was not affected. The common duct was small and healthy, so was the pancreatic duct. The whole length of the diseased part of the duct was about an inch. The microscope showed only fibrous scar-tissue in the thickening. . . . The stricture, which was situated in the walls of the hepatic duct nearly two inches above its point of junction with the cystic duct, appeared to be composed of simple connective tissue, and resembled the condition seen in an ordinary stricture of the urethra.' Here then we have clearly a case of jaundice, notwithstanding, as is proved by the colourless mucus in the gall-bladder and distended bile-ducts, that the liver's biliary function was not only totally suppressed, but had been so for a considerable time before the death of the patient.

As those of my readers who are insufficiently acquainted with the normal physiological functions of the bile-ducts may be at a loss to comprehend how the fact of a white mucous secretion instead of a dark green biliary one being found in all the ducts behind the seat of obstruction in the above case is of itself conclusive evidence that the jaundice must of necessity have been due to the non-secretion of bile, I shall briefly explain the true nature and the origin in such cases of this white mucous secretion.

Bile-ducts are mere passive canals, along which the biliary secretion flows. They take no direct part

either in the secretion or in the elimination of the bile. Their functions being passive, they have only physiologically to do with their own maintenance and integrity. Every bile-duct has a mucous membrane of its own, which secretes a white viscid mucus, which serves the purpose of lubricating its free surface, and protecting it from the chemical action of the bile flowing along the duct. The secretion of mucus by the mucous membrane of the bile-duct being perfectly independent of, or upon, the biliary function of the liver, goes on in its usual course from day to day, from hour to hour, and from minute to minute, whether or no bile be flowing along, remaining stagnant in, or entirely absent from, the duct. The only difference in these three sets of cases being—

(*a*) When the bile is flowing along the duct, the excess of mucus is carried away with it.

(*b*) When the bile is stagnant in the duct, the excess of mucous secretion mingles with the bile, imbibes its yellow colour, and remains with it stagnant in the duct.

(*c*) When there is no bile in the duct, the excess of mucus, which is now not bile-stained, accumulates there until there is such an accumulation as necessitates its flowing by itself along the duct, and, if the outlet of the duct be not impeded, it finds its way out of the liver along the common bile-duct into the intestines.

Now comes the point. What happens when the biliary function is totally suppressed and no bile whatever finds its way into the bile-ducts, and the mucus is yet secreted, but remains pent up within the ducts from an impediment in the course of the duct stopping its getting out of the liver? Simply this. The mucus gradually accumulates in, and distends, the bile-ducts in precisely the same way as bile does when it is secreted, and its exit is prevented by a similar kind of obstruction. The anatomical conditions of the ducts being identical; their contents alone are different. In the one case being white mucus, in the other dark green bile.

Now, after having proved so much, it still remains for me, in order to clinch my argument, to adduce a precisely opposite case, and show that in what is called a case of jaundice from obstruction there is not only no white mucus whatever in the biliary passages, as occurs in the parallel case from suppression, but that, notwithstanding that all the other signs and symptoms of jaundice are identical in the two sets of cases, the bile-passages are, in this instance, entirely filled with pure bile and not with pure mucus as in the other. I shall, I find, be forced to go into this point all the more fully, as even Dr. Moxon in his otherwise admirable report of his case has unfortunately fallen into the error of saying that he thinks his case 'shows that in *obstructive* [I italicised the word, G. H.] jaundice

the yellowness [of the skin, G. H.] is caused by the suppression of the secretion, and not by the reabsorption of the secreted and pent-up bile;' for while believing 'that the reabsorption occurs in cases of obstruction, he says he thinks that the reabsorption is only an unimportant accompanying incident of the early stages of the jaundice, and only concerns the bile already in the ducts, whereas the true cause of obstructive jaundice is suppression of the secretion, not its reabsorption.' Indeed he 'thinks that we may deny that reabsorption of bile is a cause of jaundice.' This is a view that I am sorry to say I cannot endorse, for, as will be shown in the sequel, it is not only contrary to modern physiological data, but at direct variance with well-established pathological facts. While thus dissenting from the views Dr. Moxon has promulgated regarding the pathology of obstructive jaundice in the latter part of his paper, I must not omit to congratulate him sincerely on the able comments he has made regarding the pathology of his case as a typical example of jaundice from suppression of the biliary function; and I do so, of course, all the more readily from the fact that they perfectly accord in every particular with the views I take of it myself. For example, he calls special attention to what he justly considers the remarkable fact that the contents of the distended bile-ducts 'were perfectly clear and colourless,' and that 'there

was no bile pigment in them at all,' while on the other hand the serum of the blood was of an 'intense golden yellow.' To this remark he appends the pertinent question, 'How is it possible that the bile which gave the serum deep jaundice could have been coming all the while from the ducts in which no bile whatever was present?' and moreover wisely adds: 'If we assume that the jaundice was really due to suppression of the secretion, then we have a ground for comprehension of the absence of bile from the mucus of the bile-ducts.' In order, then, that there may no longer exist in the mind of a single reader the remotest shadow of a doubt regarding the rationale of a case of jaundice from obstruction, before citing the other promised crucial example, from an equally independent observer, as a standard of comparison with Dr. Moxon's case, I shall first give an illustrative typical example of jaundice from obstruction in which all the morbid anatomical conditions were carefully and crucially shown, after death, to be directly opposed to those occurring in a case of jaundice the result of suppression. As it is not in the least degree difficult to find cases of this kind of jaundice, although it is very seldom that any single one of them is recorded with sufficient explicitness to be able in itself to answer the purpose of furnishing incontrovertible evidence of the exactitude of the theory on which they are based, I shall quote one from my own practice. Which,

however, from the fact of its not being unusual in any way except as regards the manner in which it was recorded, and from its having been reported in full nineteen years ago—that is to say long before I had any idea of making use of it in this controversy—cannot possibly be suspected of having its details trimmed to suit the theory. While it further possesses the immense advantage of being accompanied by a chromo-lithograph representing the morbid conditions so plainly that it cannot fail to carry along with it conviction to the mind of even the most sceptical and thereby pave the way to the easy comprehension of the parallel case of jaundice from obstruction which I have to place in juxtaposition with that from suppression recorded by Dr. Moxon. Which, like it, I select from cases recorded by independent observers, in order that it may be entirely free from the possibility of exciting in the mind of the most ungenerous reader the barest suspicion of having a theoretical colouring given to its pathological data. The typical illustrative case of jaundice from obstruction which I now proceed to lay before the reader is one I frequently saw during the lifetime of the patient along with Dr. Prance of Hampstead.

When the gentleman, who was aged about 50, was first brought to me, I was told that he had already been the subject of intense jaundice for several months. At the time I saw him his skin was

Plate I.

Mintern Bros imp

Jaundice from Complete Occlusion of the Orifice of the Common Bile - Duct.

a Intra-parenchymatous ducts b Enlarged Hepatic duct c. Enlarged Cystic duct d Enlarged Common Bile - duct
e Distended Gall - bladder f Cicatrised Duodenal Ulcer at the orifice of Bile duct g Dilated Pancreatic duct

indeed so intensely discoloured with bile-pigment that he looked not yellow, but of a dirty yellowish green. His fæces were said to be absolutely devoid of bile, having been for months past of a light pipeclay colour, while his urine was of a dark—very dark—saffron hue. In fact, he was a typical case of jaundice, from every single point of view. At first the liver-dulness had been increased, and then, as in all other cases of jaundice from obstruction, it shrank down until it resumed its normal dimensions. But unfortunately the shrinking process did not stop here; for, in consequence of the still continuing arrest to the outflow of the bile, the backward pressure on the secreting structure was continued, and as a natural consequence the liver became smaller and smaller until an actual atrophy of the gland was the result. The patient ultimately died, and the condition of things admirably represented by Mr. Ford in the chromolithograph Plate I. was found at the autopsy.

1. The liver was small and deeply stained with bile-pigment, being actually of a blackish-green colour. Not alone from the bile having been long retained in the organ, but from its having permeated every cell, every fibre, and every granule of the tissues composing the parenchyma, ducts, and vessels of the liver.

2. Every bile-duct was greatly dilated—not only the cystic, hepatic, and common bile-ducts, but even

those in the tissues of the liver themselves, as is shown on a reduced scale in the plate at *a*.

3. The common bile-duct (*d*) was distended to eight or ten times its natural size, and the hepatic (*b*) and cystic (*c*) ducts were also greatly dilated, though not in like proportion.

4. The gall-bladder was at least fifteen times its natural cubic size (*e*).

5. All the bile-passages, as well as the gall-bladder, were filled cram-full with dark, tarry, thick viscid bile.

6. The whole of this mischief was found to be due to a permanent occlusion of the duodenal orifice of the common bile-duct by a cicatrised ulcer (*f*) preventing the flow of the secreted bile into the intestines, and the consequent penning of it up in the organ itself.

7. The jaundiced condition of the skin, &c., was due not to an arrest of secretion—for, as was shown by the loaded state of the gall-bladder and ducts, plenty of bile had been secreted—but to a reabsorption of bile from the gall-bladder and bile-ducts by a process of capillary osmosis.[1]

Here then is what may be considered to be a typical case of jaundice from obstruction, which, I think, will be found to resemble very closely, in all

[1] Further scientific details of this important case will be found in the chapter devoted to the chemistry of the excretions, p. 769.

its salient points, the crucial case which I am now about to cite from the practice of an independent observer, in order that it may be compared with Dr. Moxon's case of jaundice from suppression.

This case of obstruction is one that was reported by Mr. John H. Morgan in vol. xxvii. of the Pathological Society's 'Transactions,' at page 176, under the title of 'Enormous Dilatation of the Bile-ducts from Stricture of the Ductus communis choledochus.' It strikingly resembles Dr. Moxon's case in the three following salient particulars. Firstly, the jaundice was intense. Secondly, the ducts were dilated to such an extent that their blind extremities looked like cysts projecting from the surface of the liver. Thirdly, the obstruction was the result of a stricture caused by inflammatory thickening.

The difference in the two cases again was—that the ducts, instead of being filled (as in Dr. Moxon's case) by a clear white mucus, were choke-full of dark black inspissated bile. The case is briefly as follows :—The patient, a man aged 52, by occupation a plumber, died intensely jaundiced in St. George's Hospital. He had enjoyed good health until four months before his admission, when, after receiving a prick in the hand, which was followed by an abscess, he was seized with shiverings and diarrhœa, and a day or two later became jaundiced. On admission the skin was bright yellow, irides and conjunctivæ being deeply

stained. The stools light stone-coloured, and the urine loaded with bile. There was pain at the right side of the ensiform cartilage, but no enlargement of the liver. In a month he began to suffer from such intolerable itching that he scratched his skin till it bled. A couple of weeks later the liver was found to have become enlarged. Both lobes projected so much forwards as to look like two tumours slightly elevated above the surrounding surface of the body. The patient died about ten weeks after his entrance into the hospital, and at the post-mortem the following condition of the liver was observed. It 'was greatly enlarged and distended; its surface smooth and presenting several slight elevations, whose thin and transparent walls and fluid contents had all the appearance, at first sight, of cysts. These were found especially on the under surface of the left lobe, their sizes varying from an inch and a quarter to a quarter of an inch in circumference. The gall-bladder was very much distended; it measured from five to six inches, and projected some distance below the edge of the liver; it contained dark green inspissated bile mixed with mucus and epithelium. A stricture of the common choledic duct existed just below the point of its formation by the union of the cystic and hepatic ducts.' The contraction of lymph arising from the presence of a perforating ulcer of the duodenum (bound down by adhesive bands of lymph to the

bile-duct) was found to be the cause of the stricture. This obstruction to the outflow of the biliary secretion 'had caused the distension of the gall-bladder and its duct, and also those of the liver, which it had distended to such an extent as to cause their extremities to project on the surface as above described.'

Here then is a case of jaundice from obstruction to the outflow of bile, parallel in every respect, as regards its morbid anatomy, to that of Dr. Moxon's of jaundice from suppression of the secretion. In the one case the stricture of the duct being the cause of the jaundice, while in the other case the jaundice *was quite independent of the stricture of the duct*, as is proved—and indubitably proved—by the fact that behind the seat of stricture mucus alone, and no bile whatever, was found pent up in the occluded duct.

I now leave it for the reader to draw for himself a mental comparison between these typical examples of the two distinct and independent forms of jaundice, and form his own conclusions as to the justice of dividing cases of jaundice into the two great classes of jaundice from suppression and jaundice from obstruction. To my own mind the thing is as clear as noonday; for the mere fact of the secretion pent up in the hepatic bile-ducts being dark bile in the one case and pale white mucus in the other sets at once and for ever aside all possibility of reasonable discus-

sion against the theory of jaundice from suppression, and raises the statement from the humble position of a mere theory—which it must be admitted, scientifically speaking, it has hitherto only occupied—to the dignity of an unassailable and imperishable clinical as well as pathological fact. Deny it who can! Should, however, any reader be so unfortunate as to have failed to grasp the intrinsic value of the data upon which the theory of jaundice from suppression is founded, which is the most difficult one for the non-scientifically trained mind to understand, I will ask him to turn to page 1083, and carefully read the chapter on gall-bladders distended with mucus, as well as give attention to Wyss and Ritter's observations at pages 708 and 1088 ; and if his eyes are not then opened, I am sorry to say I can do nothing more for him, but leave his case to time and the developmental powers of nature.

Only one thing further will I allude to—namely, the fact, which is strongly corroborative of my views, that the serum of the blood in Dr. Moxon's case was —like the skin—deeply stained with yellow pigment, notwithstanding that the liver was secreting no bile, and the urine loaded with dark pigmentary matter, exactly as occurred in my case of jaundice from obstruction, about the pathology of which there can exist not a shadow of doubt.

The condition of the blood in Dr. Moxon's case

is a fact which of itself distinctly corroborates my other belief that biliverdin is nothing more or less than oxidised blood hæmatin, which the liver does not form, but only extracts from the circulation during the passage of the blood through the hepatic capillaries. The natural yellow colour of blood serum being due to the pigment which, when extracted from the circulation by the liver, we call biliverdin, or bile-pigment.

When biliverdin is regarded in this light, it greatly simplifies the comprehension of all the various widely differing causes of jaundice. Thus, it shows us that the jaundice of snake poison may be due to the transforming power the venom germs have upon the oxidation of the blood hæmatin. While again as regards the cause of the yellow discoloration of the skin in *febris icterodes*, as well as in *febris icterodes remittens*, if we cease to look upon them as peculiar species of malignant fevers, accidentally associated with a jaundiced condition of the skin, and adopt the view, which I espouse, that they are in reality mere disorders of the hepatic organ due to the introduction into the system of toxic agents in the shape of disease-germs, which cause jaundice from suppression, their comprehension becomes simple enough. For, as I shall presently show, not only animal and vegetable, but even mineral poisons, have the power of suspending the secretion of bile and giving rise to jaundice.

Moreover, the more closely we examine the so-called yellow fevers the more evident does it become that they differ in no respect whatever from the diseases arising in temperate climates from the introduction of animal and vegetable disease-germs. Except in so far as the *febris icterodes* is highly contagious, and that both it and the *febris icterodes remittens* only originate in tropical climates.

I have still some further observations to make on the mode of the production of the forms of jaundice now under consideration; but as what I have to say is equally applicable to all the forms of jaundice arising from the introduction into the human system of poisons, no matter whether they be animal, vegetable, or mineral, I shall delay my further remarks on the subject until after I speak of jaundice the result of acute atrophy of the liver, which I regard as being due to the introduction into the system of a toxic agent (page 391), for the rationale of all the poison cases is, in this respect at least, identical.

The next point in connection with the etiology of jaundice requiring solution is the question: Why does bile-pigment select the rete mucosum of the skin as the chief seat of its deposition? This is a question which almost every intelligent practitioner, as well as every pathologist, must have put to himself. I question, however, if even so much as one in fifty has ever been able to find a satisfactory answer to his

mental query. Indeed, as far as I am aware, not a single writer on jaundice, either separately or in conjunction with cases of liver disease in general, has ever so much as attempted to explain the etiology of the deposition of bile-pigment in the rete mucosum of the skin, although, no doubt, every one of them must have thought the matter over. I know I have often done so, and as I imagine that I have found a good, if not even the correct, answer to the query, I shall venture to lay it before my readers, and allow them to judge for themselves as to its merits. If they do not consider it satisfactory, and if any one among them thinks he can give a better, I shall be delighted if he will kindly communicate it to me.

Having already, I think, made it sufficiently clear that the yellow pigment deposited in the rete mucosum in all cases of jaundice must have originally come from the blood, I have now to show that while, in jaundice the result of suppression, the skin derives it directly and at once from that original source, in jaundice from obstruction the pigment has not only been previously extracted from the blood by the secreting cells of the liver, but, after having been pent up along with the other constituents of the biliary secretion in the hepatic ducts and gall-bladder, again been absorbed back from thence by a process of capillary osmosis into the general circulation, before

being ultimately deposited in the rete mucosum during the attempt made by the skin to eliminate it from the body.

The latter part of this last sentence embodies the key to the solution of the question, Why does the skin become yellow? The answer, in my mind, is very simple. The kidneys and the skin are the two great eliminating emunctories of foreign non-volatile matters from the body, both in health and in disease, acting not only conjointly but vicariously in this respect, and the bile-pigment is eliminated by them.

In case some reader may have for the moment forgotten the immense eliminating power of the skin, I may remind him that when in disease the excretory functions of the kidneys are arrested, the sweat and sebaceous glands of the skin immediately vicariously assume their office, and not only eliminate the soluble salts of the urine—such as urea, uric acid, chloride of sodium, and sulphate of potash—but even the insoluble, such as the oxalate of lime; and that sometimes to such an extent, that the whole surface of the skin, on becoming dry after a profuse sweating, is covered with a rough white crystalline powder of oxalate of lime, just as the ground is with hoar-frost in a sharp cold spring morning.[1]

[1] See Author's work on the Urine. English, American, French, or Italian editions.

In health it is the kidneys alone which eliminate all the effete pigments—hæmatin, biliverdin, &c.—in the form of urohæmatin (urine pigment); but in disease the skin takes a share in this office. Hence, when the blood-serum is loaded, as in jaundice, with pigmentary matter, the skin as well as the kidneys excrete it, and the perspiration, like the urine, becomes so loaded with it that white linen, brought in contact with either the one or the other, becomes dyed of a deep yellow hue. Now having arrived at this point in the argument, we have only to go one step further, and our journey is at an end. Look at Plate II. p. 728. There is shown a kidney which for months has been occupied in eliminating bile-pigment, and, notwithstanding that the so doing is one of its normal functions, it has broken down under the abnormal strain, and as a consequence visible masses of non-excreted bile-pigment are seen scattered all over its external surface. A precisely similar state of matters was observed in its interior. Now what happened in this case with the skin? It not being the *normal* function of the skin to eliminate bile or any other animal pigment, but this work having been abnormally forced upon it, and it being compelled to perform it to the best of its ability—which after all it can do but imperfectly—a great part of the insoluble pigmentary matter remained behind in the rete mucosum, thereby giving to

the skin the yellow hue which we call jaundice. Which is not in the least to be wondered at, seeing that the skin, from possessing no special apparatus for eliminating the bile-pigment, does nothing more than, as shown in Plate II., the kidneys themselves do when an excess of work is thrown upon them—does the work imperfectly, breaks down, and, instead of passing all the bile-pigment through its cells, allows a quantity of it to remain behind in the rete mucosum, which, by gradually increasing in quantity, causes the skin to assume by degrees a deeper and deeper jaundiced hue, until the patient may at length become of an almost greenish-black tint.

From this theory it is seen that I entirely ignore the idea that the cells of the rete mucosum store up the pigment from any choice of selection of their own, but, on the contrary, as the inevitable consequence of a dire necessity. The cuticular cells being, like the kidney shown in the plate, unable to eliminate all the bile-pigmentary matter brought to them by the circulation, become filled with it *nolentes volentes*, and are thereby made to assume the colour which we denominate jaundice.

It may perhaps be as well for me here to call attention to the fact that the pathological conditions included under the name of jaundice are not peculiar, or limited, to the human being. For, on the contrary, all animals—fish and fowls as well as qua-

drupeds—are liable to be attacked with precisely the same pathological conditions. I have myself seen jaundice in a domestic fowl, a sheep, and an ox. Indeed, one of my own horses had on one occasion a very smart attack of jaundice. The reason why this condition appears to us to be rare in our domestic animals arises from the fact that the abnormal tint of the skin is hidden from us in consequence of the animals possessing a thick covering of feathers or hair, and of the conjunctiva from their eyes showing little or no white. Consequently, it is only by examining the colour of the tongue and inside of the lips of such animals that the jaundiced condition of their bodies can be detected during life. The effusion of biliary pigment into the buccal mucous membrane of my own horse, which was attacked with jaundice, was most marked, and at once furnished me with the explanation of the fact that patients suffering from jaundice had again and again complained to me of having a disagreeable bitter taste of gall in their mouths.

CHAPTER IV.

SIGNS AND SYMPTOMS OF HEPATIC DISEASE.

Although the signs and symptoms will be given fully and in detail when each individual disease of the liver is being specially considered, it will be well for me, in order to make their intrinsic diagnostic value more easily understood, to give a brief general view of the more important of them before entering upon the consideration of each disease separately. So I shall do so now.

In case some of my younger readers may marvel at my heading this chapter *Signs and Symptoms*, I may as well remind them that a sign is a very different thing from a symptom, and has a totally different diagnostic value. For while a sign is something that the medical practitioner can himself see, feel, or hear, with his own eyes, fingers, or ears, a symptom is something of which the patient is alone physically conscious, and consequently the medical attendant's knowledge of its existence and nature is derived solely from the patient's account

of it. Hence the value of a symptom in a great measure depends upon the mental capacity as well as the veracity of the patient. So that there is an unmistakable difference to be attached to the intrinsic diagnostic, prognostic, and therapeutic clinical value of signs and symptoms, of each of which I shall now speak separately.

The Tongue.—The condition of the tongue is oftentimes very characteristic of liver disease. Every bilious patient, without exception, has a foul tongue. Always white, often furred, frequently yellow, especially at the back, where the yellowness frequently ends in a brownish tint.

In abscess of the liver the tongue is said to be scarlet, or what is commonly called raw-flesh looking, anteriorly, and furred posteriorly. But every case with a raw-flesh looking tongue is not to be diagnosed as one of suppuration of the hepatic tissues ; for raw-flesh looking tongues are not unfrequent in simply inflammatory conditions of the liver.

The Taste.—A bitter taste is commonly complained of in many forms of liver disease, and this is owing to the fact that in them the blood becomes surcharged with taurocholic acid—the substance which gives to bile its intensely bitter flavour—which is brought into contact with the papillæ of the tongue by the buccal capillaries.

The Bowels.—The condition of the bowels is variable in liver disease. They are confined or open in exact proportion to the quantity of bile excreted. When, for example, no bile finds its way into the intestinal canal, as in all the various forms of obstruction to the outflow of bile from the common bile-duct, constipation is an inevitable consequence. While, on the other hand, when there is an excessive excretion of bile into the intestinal canal, a diarrhœa is the result, from bile possessing among other of its attributes purgative properties—inasmuch as it favours intestinal peristaltic action—which, by older writers, was described, and is still spoken of among the laity, as 'a bilious diarrhœa.' In such cases, however, there appears to be something further wrong than a mere hyperexcretion of ordinary bile; for the stools are not only dark in colour, but profusely charged with intestinal mucus. Which fact proves either that the biliary secretion must be unusually irritating, or that there exists at the same time an inflammatory condition of the intestinal canal to induce the hypersecretion of mucus; an unusually irritating condition of the bile being the most likely cause.

a. Pipeclay-coloured stools are due, not to the presence of any new and foreign material, but entirely and always to the absence of the normal biliary pigment. Hence they are only present in two sets of cases. Firstly, when there is a suspen-

sion of the biliary secretion. Secondly, when there is an obstruction to the flow of the secreted bile into the intestinal canal.

b. Black, tarry, bilious-looking stools may exist when not one drop of bile finds its way into the intestinal canal. Which circumstance has frequently given rise to grave errors in diagnosis. Tarry, bilious-looking stools are, for example, met with when the biliary function is totally suspended, as in some cases of acute atrophy of the liver, as well as in cases of contagious jaundice—commonly called yellow fever. In both of these sets of cases, the black, tarry, bilious-looking stools are due to the presence of blood in the fæcal matter.

Hæmorrhage from the bowels not only occurs in many acute forms of liver disease—especially those in which the liver is softened—but also in exactly the opposite condition of things, namely, in chronic diseases where the liver is hardened. As for example in cirrhosis, and all other forms of hepatic disease which impede the portal circulation. A precisely similar cause induces the formation of internal piles, the bleeding from which is often profuse in advanced cases of cirrhosis. Hæmorrhage of the bowels is also frequently met with in cases where a gall-stone ulcerates its way into the intestines, from its opening one or more of the intestinal blood-vessels.

The presence of black stools, when no bile enters the intestinal canal, may also be due to foods and remedies.

Flatulency.—The intestinal flatulency which is not only a common, but oftentimes a most distressing, symptom of liver disease, is readily accounted for from bile being an anti-putrefactive in intestinal digestion. When bile is either entirely absent, as it frequently is in certain forms of liver disease, or present in insufficient quantity to admit of the intestinal digestion proceeding in a normal manner, the contents of the intestinal canal undergo putrid fermentation, and large quantities of foul-smelling gases are disengaged, which, not being absorbed into the circulation, to be eliminated by the lungs, are expelled from the intestinal canal, upwards and downwards, to the great inconvenience and discomfort of the patient.

The Urine.—The kidneys, from being the chief organs which act vicariously with the liver, have their secretions more or less affected in all the various forms of hepatic disease.

In the inflammatory and febrile classes of liver affections, the urine is not only scanty and high-coloured, but loaded with brown, yellow, pink, or vermilion-coloured lithates.

In cases of atrophy it contains two abnormal substances, named respectively tyrosin and leucin. In

chronic congestions it contains a superabundance of oxalates as well as urates. In cancer there is usually an excess of uric acid. In all cases of jaundice the urine becomes of a saffron colour, from containing an excess of bile pigment; while in those cases which result from an obstruction to the outflow of bile into the intestines, bile acids are detectible in it. When sugar appears in the urine during the course of chronic hepatic disease, it is usually the precursor of afatal termination.

Urinary calculi—even those consisting of cystin and hypoxanthin—are in a great measure due to disordered hepatic function.

The Pulse.—The pulse in all acute forms of liver disease is rapid. In almost all the sluggish and chronic, normal, or even slower than natural.

The Temperature is high in the acute, and low in the chronic, forms of liver disease. Even in the ordinary run of minor cases of derangement of the liver's function, patients often complain of feeling chilly; and this is easily accounted for on the supposition of the calorifying function of the liver being in general, at the same time as its biliary one, out of order.

Blindness.—Defective vision is common in all cases of suppression of the biliary function. The biliary matters circulating in the blood, acting as toxic agents on the nervous system, give rise to disordered vision, muscæ volitantes, &c.

Vertigo.—Vertigo is a symptom familiar to all bilious individuals. I have seen persons holding on to the furniture of a room while crossing it when labouring under a bilious attack, in order to prevent themselves falling upon the floor. The swimming in the head or giddiness being so great, that they could neither walk straight nor stand steady.

Headache.—Headache is a usual accompaniment of liver affections, a 'bilious headache' being a common occurrence. The pain is usually frontal, and chiefly over the eyes. Sometimes, however, it is not only behind the ears, but actually occipital.

Hepatic Pain.—Continuous and acute pain in the region of the liver is common in all the various forms of inflammation, whether it be merely congestive or suppurative. Continuous dull ache is the accompaniment of all forms of tumour of the liver, except one; and that is hydatid tumour, which is as a rule painless. Acute paroxysmal pain occurs where there is a gall-stone, inspissated bile, an entozoon, or some other foreign body impacted in one of the bile-ducts.

Shoulder Pain.—A great deal was formerly written and said regarding the diagnostic value of right shoulder pain in cases of hepatic disease. It was learnedly accounted for by the supposed anastomosis of a twig of the supra-clavicular nerve with the right pneumogastric. In the early part of my career, I, like most other people, paid great attention to it, never by

any chance omitting to ask every patient who came to me labouring under liver disease if she or he had or had not shoulder pain ; but the conclusion I have arrived at is, that shoulder pain is an utterly worthless symptom, being frequently absent when it ought, if the theory were correct, to be present, and still oftener present when the liver is not affected at all.

Another 'idea' has been recently started by Dr. Vidal ('Progrès Médical,' 1879), and that is, that, in affections of the liver, there is a painful spot at the spinous process of the fourth dorsal vertebra ; that, in perityphlitis, patients have a painful point at the junction of the second and third dorsal vertebræ on the left side ; and that, in ulcer of the stomach, the patient has commonly a painful spot over the spinous process of the sixth dorsal vertebra. Time will show whether this idea of Dr. Vidal's is anything more than 'an idea.' Meanwhile, as I have not yet put it sufficiently to the crucial test of experience, I will say nothing either for or against it, but leave my readers to make what use of the hypothesis they please.

Hepatic Neuralgia.—It is common to find in the works of non-scientific physicians a condition of liver spoken of under the title of 'Hepatic Neuralgia.' Of course a man who is guilty of describing hepatic pain as a pathological diseased state must necessarily be a believer in 'symptom diseases.' So it naturally

happens that the man who writes learnedly on the one is invariably found to speak equally learnedly of the other. To my way of thinking, the so-called hepatic neuralgia is nothing more or less than ordinary pain, the direct result of hepatitis, cancer, gall-stones, inspissated bile, or some other equally physical disordered condition of the parenchyma of the liver or its ducts.

Hepatic Amenorrhœa.—This so-called hepatic symptom, which one occasionally sees spoken of in works on 'Diseases of Women,' appears to me to be little less logical than the hepatic neuralgia treated of in books on diseases of the liver. For after having given considerable attention to the matter, the conclusion I have arrived at is that the effect of liver disease on menstruation is both doubtful and indirect. For example, we hear people speaking of amenorrhœa as a consequence of fatty liver; but as amenorrhœa is a common concomitant of many other diseased conditions of the body, I see no reason why a fatty liver should be put specially forward as an exciting cause of amenorrhœa. Is amenorrhœa not common in phthisis? and phthisis, when treated with cod-liver oil, is often associated (as I have shown at page 1017) with a fatty condition of the liver. Why then in such cases should not the phthisis itself bear all the blame of the amenorrhœa? Again, it is not uncommon to hear obstetricians glibly talking of a congested state

of the liver as being the *direct result of amenorrhœa.* Apparently for no other reason than their thinking that as there is no monthly flow of blood from the uterus, and the patient's liver chances to be congested, it is not only possibly but probably so. Too much blood being in the body, from none being passed by menstruation, and not being very particular as to which organ it elects to locate itself in, it does so in the liver—probably, one might suppose, from that organ being the most capacious, and at the same time being situated not very far distant from the uterus ! If persons of this pathological turn of mind would only consider for a single moment the morbid anatomical conditions of the blood-vessels which are essential to the production of a hyperæmia, or congestion as it is commonly called, in the liver or any other organ, they would not be quite so ready to give utterance to such theories. For although it is an undeniable fact that fatty liver and amenorrhœa, as well as congested liver and amenorrhœa, do oftentimes exist simultaneously in the same individual, it by no means follows that they stand in the relation to each other of cause and effect. In fact there are cases which would indicate quite the reverse state of matters. For profuse menorrhagia occasionally occurs in patients under treatment for chronic congestion of the liver. I am glad to see that Dr. Matthews Duncan has apparently eman-

cipated himself from this erroneous obstetrical theory. For I find that in a lecture of his on hepatic disease in 'Gynæcology' (published in the 'Medical Times and Gazette' of January 18, 1879) he refers to the case of a woman, 31 years of age, who was treated in St. Bartholomew's Hospital for menorrhagia the direct result of congestion of the liver, described in the lecture as a case of chronic hepatitis.

The Skin.—From the circumstance of the skin, like the kidneys, acting vicariously with the liver, its functions are always more or less deranged in hepatic disease, and the form of derangement most commonly assumed, as well as the one which is most visible to the eye, is jaundice. However, jaundice is not invariably present in all liver affections. Indeed, so frequently is it totally absent that I have given this book the title of 'Liver Diseases with and without Jaundice.' As it is, however, much more frequently present than absent, and moreover is never present except when the liver is diseased, it behoves me here to speak of this sign very fully. The peculiar yellow tint of the skin which is so characteristic of certain forms of liver disease received from the French the vernacular name of 'jaunisse,' and was corrupted by bad English pronunciation and still worse English spelling into 'jaundice.'

The vernacular English word for the affection is 'yellows.'

Although it requires no medical training whatever to distinguish by the colour of the skin the existence of this morbid state, it will be nevertheless necessary for me to go minutely into the details of several other signs and symptoms connected therewith, as they are not always at a glance palpable even to the medically trained mind. They are the following :—

A. All forms of jaundice are invariably accompanied by saffron, greenish, or blackish-coloured urine, which stains the linen from a pale golden yellow to a deep orange tint.

B. The stools are usually pipeclay-coloured, constipated, and very fœtid. When the stools are dark in a case of jaundice, it arises from blood having escaped into the intestinal canal.

C. The perspiration is saffron-coloured, and stains the linen of a rich golden-yellow hue.

D. The tongue is foul, and the appetite bad.

E. The temperature of the body in cases of jaundice is never increased, except when the disease is associated with pyrexia. In fact it is usually diminished, especially in all the various chronic forms of the disease depending upon obstruction of the common bile-duct.

F. The pulse follows the temperature ; being quick when it is high, slow when it is low.

G. Occasionally, though exceedingly rarely, a

condition of yellow vision is present which has received the technical name of 'xanthopy'—*xanthos* being merely the Greek word for 'yellow;' the English word not having been considered by some who bow down and worship grand names good enough for it.

Yellow vision, let it be remembered, is a much more common symptom of the toxic effects of santonin on the system than of jaundice; and as santonin also makes the urine yellow, care must be taken not to make a mistake, by jumping to a conclusion before thoroughly investigating the case.

H. Disordered cerebral symptoms, such as delirium, convulsions, and coma, are present in the worst forms of the affection. More especially in cases of contagious jaundice and acute yellow atrophy of the liver, as well as in those of sudden suspension of the biliary function from enervation, induced by mental emotion and animal, vegetable, and mineral toxic agents. (See page 413.)

I. Whenever bile ceases to find its way into the intestines, great disorder in the digestion and absorption of the food takes place. Not only does a portion of the fatty foods cease to be emulsioned and absorbed, but a form of putrid fermentation is set up, and a copious disengagement of foul-smelling gases is the result, causing great distress to the patient, from both the fæces and flatus passed having a most noxious odour.

K. The skin does not, immediately after bile ceases to find its way into the intestines, assume a jaundiced tint. Sometimes not even for three, four, or six days. Though generally within seventy hours a distinctly lemon tint begins to be visible.

L. The conjunctivæ and skin become yellow almost simultaneously, but the conjunctivæ usually first.

M. The urine becomes saffron-coloured sooner than the skin gets yellow. Usually within thirty-six hours after the flow of bile into the intestines has ceased.

N. In some very rare cases,—like the extraordinary one related by Bleicher in Schmidt's 'Jahrbuch,' p. 48, 1839—the buccal mucous membrane has been noticed to be blue instead of being, as it usually is, yellow. Exactly in the same way as the urine has been observed to be of a similar tint in cases of jaundice, as well as of other diseases. This arises from a mere modification of the degree of oxidation in the animal biliary pigmentary matter having occurred from some accidental constitutional complication of the system. Bile pigment, like urohæmatin, may be oxidised of a yellow, green, blue, purple, or red colour.

N.B.—When the common bile-duct is ligatured in animals, a yellow condition of the buccal mucous membrane begins to make its appearance, as a

general rule, within seventy-two hours after the operation, but a distinct jaundiced condition is not usually observable before the eighth day after the application of the ligature. The urine and stools show the effects of the ligature much sooner than the buccal mucous membrane.

O. In all cases of jaundice, the bile pigment is found to stain different parts of the body with varying degrees of intensity. The skin of the abdomen is usually the most intensely stained, and next that of the face. Of the internal organs, the liver in jaundice from obstruction is by far the darkest part of the whole body, amounting sometimes even to a greenish-black. All the parts in contact with the gall-bladder and external bile-ducts are also intensely stained. The fat over the whole body is likewise deeply tinted.

P. Sometimes jaundice (like sweating) has been found limited to one half of the body. Dr. Frank relates a case of this kind, in the 'Prax. Med. Univ. Præcept.,' 1843, under the title of *Icterus Dimidiatus*. Their pathology is, in my mind, perfectly explicable from the cutaneous excretion of the bile pigment being under the influence of the nervous system, just as the perspiration is. Unilateral jaundice, therefore, is the result of the excretory nerves (not necessarily at the same time either the motor or sensory) of one side of the body being in a state of hemiplegia.

In 1853–4, I saw a strange case of 'hemi-sweating' in a man in the clinical wards of the Würzburg Hospital, which at once suggested to my mind the theory of 'hemiplegic paralysis of the excretory nerves;' and if that theory were true in the case of the dimidian sweating, I think it may be equally true in that of dimidian jaundice.

Q. Besides the urine and sweat, other normal secretions—such, for example, as the saliva, milk, and even the tears—have sometimes been found to be of a yellow hue in cases of marked jaundice. Legg found the serum, both in the chest and body after death, even in cases of slight jaundice, give an intense bile reaction with nitric acid, even when the urine gave scarcely any. Not only have I seen the serum in cases of jaundice with ascites so deeply tinted as to stain linen yellow, but I have seen the serum of blood drawn from jaundiced patients do exactly the same thing, and stain white paper of a deep yellow or saffron hue.

Having said that the serum of the blood may be of a deep yellow colour in jaundice from obstruction, I ought to remind the reader that it was pointed out at page 111 that the serum of the blood in cases of jaundice from suppression is equally of a deep bilious yellow hue, which of course arises from the simple fact that bile pigment is one of the biliary products not manufactured by the liver, but only

secreted by it, already formed from the blood. For, as before said, bile pigment is merely blood hæmatin in a different stage of oxidation.

R. There is an occasional accompaniment of jaundice—it cannot be called a sign of the disease—which has received the title of 'xanthoma,' or 'xanthelasma,' from some, and 'vitiligoidea' from others; which consists of a tumefaction and discoloration of the skin in particular parts of the body, more particularly the eyelids, the scrotum, and the hands, of which I shall afterwards, at page 1061, more particularly speak.

S. Pruritus, shingles, and nettle-rash are not uncommon concomitants of disordered biliary function, and intense itchiness of the skin, without any eruption whatever, is oftentimes the most distressing symptom the patient complains of in cases of jaundice. It is usually worst at night, when the patient first goes to bed, and sometimes almost amounts to the 'torment of the damned,' leading patients to lacerate the skin with their nails till it literally streams with blood. So intense is the itchiness, that sometimes strong anodynes, both by mouth and by subcutaneous injection, fail to give relief.

N.B.—Acid baths increase the irritation, while alkaline baths slightly relieve it. The best treatment of all is to give half a teaspoonful of bicarbonate of soda and from two to ten grains of the

iodide of potassium in six ounces of water, just before going to bed. This I give on physiological chemical principles, believing, as I do, that the cutaneous irritation is due to the effects of the bile acids—glycocholic and taurocholic acids—circulating in the blood, and irritating the extremities of the cutaneous nerves. The addition of soda to the blood transforming these irritating acids in the body, as it does out of the body, into the comparatively speaking non-irritating salts, glycocholate and taurocholate of soda.

I am all the more convinced that the cutaneous irritation met with in jaundice is due to the bile acids, from the two following facts. First, it is always most marked in cases of obstruction of the duct, where reabsorption of the secreted bile is the invariable cause of the discoloration of the skin ; and, secondly, of all the biliary substances that I have seen injected under the skin of dogs, the bile acids have always produced the most marked symptoms of nerve irritation. (See further remarks at p. 739.)

T. During an attack of jaundice the catamenia are occasionally observed to be suppressed. And a few cases have even been reported where jaundice instead of menstruation has been observed to occur for three or four days at the monthly period. Which fact led Dr. Hirschberg to ventilate the hypo-

thesis that jaundice might in some cases be a mere vicarious form of menstruation. Those of my readers who may feel interested in this bold hypothesis will find Dr. Hirschberg's paper in the 'Reports of the Berlin Medical Society' for 1872. All that I shall remark upon the subject is merely that the menstrual function is frequently suppressed in a variety of other diseases as well as in some of those associated with jaundice, and that, bearing on the same subject, it is a matter of common observation that great losses of blood are oftentimes accompanied with a sallow complexion almost amounting to actual jaundice. Besides which :—

U. Cases of actual jaundice following upon hæmorrhage have been frequently recorded. Mr. W. Smith of Clifton relates the case of a strong and healthy collier, aged 24, who, in killing a duck, thrust the knife into his wrist in the direction of the radial artery. From the profuse and dangerous bleeding which ensued, and other circumstances, the artery was probably wounded. The hæmorrhage was excessive. The wound did well, and healed without difficulty. But, about a week after the accident, the pulse assumed a febrile rapidity, the skin became hot, and there were alternate chills and flushings with nausea and anorexia. The patient was jaundiced, and continued so for about three weeks. The skin and conjunctivæ were of a deep yellow hue, the

stools light, and the urine tinted with bile pigment. The bowels were constipated ; but, after the feverishness subsided, his general health improved. He got no active medicines, only effervescents, mineral acids, and an occasional dose of castor-oil.

He relates another case much resembling this, except that the hæmorrhage was venous, instead of arterial. A farmer, aged 56, suffered much from varicose veins. One, over which there had been an ulceration, gave way, and an immense quantity of blood was lost. The condition of this man was like that of the one before described, and the history of the case, to a certain period, the same. Well-marked febrile reaction, with jaundice, set in about the fifth day. He recovered in the same manner as the former case.

To these cases he adds one of jaundice in a lady after alarming epistaxis, and another after uterine hæmorrhage. But this last case is of doubtful value, as the patient was not attacked with the jaundice until a month after the delivery, having suffered the previous day from shivering, headache, and symptoms of approaching fever. The yellowness of skin, white stools, and bilious urine, lasted about a week ; the treatment being two grains of calomel and a dose of castor-oil every other morning, with a taraxacum and nitric acid mixture thrice daily.

I too have seen very sallow complexions more

than once after severe hæmorrhage. One very recently, where the gentleman died. But the yellow skin was due, not to biliary derangement, but to the mere loss of blood. This patient I saw along with Dr. Foakes and Mr. Parrott, and I do not suppose that either of these gentlemen, any more than myself, dreamed of calling the yellow discoloration of the skin jaundice.

Such being a brief synopsis of the signs and symptoms of jaundice, I have now to direct attention to a variety of conditions of the skin which have been, and are again likely to be, mistaken for it by the uninitiated. They are :—

1. The sallow yellow complexion of chlorotic and other bloodless patients. After having shown that actual jaundice may follow upon profuse hæmorrhage, it may be as well for me to remark that loss of blood is always followed by a sallowness of the complexion, even when no jaundice exists.

2. The sallow yellow complexion of persons who have lived long in a hot damp climate.

3. The sallow complexion of persons affected with the cancerous cachexia.

4. The sallow complexion of acute syphilis.

5. The sallow complexion of mercurialism.

6. Many new-born children have yellow skins, without any other sign or symptom of jaundice

whatever; and to this condition of spurious infantile jaundice has been given the title of 'jaundice neonatorum,' the pathology of which will be explained under the heading 'Intra-uterine Jaundice.'

7. A yellowness of the skin, closely resembling jaundice, may be produced by the intentional application of various colouring matters to the cuticle. But the deception is easily detected by the free application of soap and water, which at once reveals the true nature of the case.

8. Certain medicines have the power of making not only the skin, but also the urine yellow. This is particularly the case with the picrate of potash and santonine; and this last does so in such a marked manner that a case of this kind was once brought to me by an intelligent medical practitioner as one of jaundice. And it is not surprising that he did so; for, in spite of my familiarity with cases of the kind, it was not until I had applied the crucial test of adding liquor potassæ to the urine that I felt perfectly certain that it was not a real case of jaundice, complicated with spurious santonine cutaneous coloration.

When caustic potash is added to urine containing any vegetable colouring matter, such as rhubarb, santonine, &c., and the mixture boiled, it becomes red; whereas this is not the case with bile-pigment; it then only browns. Besides which, the addition

of strong nitric acid turns the urine green when it contains bile, while nothing of the kind occurs with yellow vegetable pigmented urine.

N.B. The crucial test for all spurious forms of jaundice is a very simple one—namely, a naked-eye inspection of the fæces. If the stools be pipeclay-coloured, the case may be at once put down as one of jaundice. If, however, the stools are dark-coloured, that circumstance of itself does not negative the idea of jaundice; for the dark colour of the stools may be due to the presence in them not of bile, but of blood. Or it may be due to medicine—for all metallic remedies whose sulphurets are of a dark colour turn black in cases of true jaundice, from the sulphuretted hydrogen disengaged during the putrid fermentation of the non-bilified food in the intestinal canal combining with the metallic remedies, such as mercury, iron, bismuth, &c., and forming black sulphurets, which, mixing with the pipeclay-coloured fæces of jaundice, give them the appearance of being coloured with bile, and lead to an error in diagnosis. Hence, before concluding in a case of yellow-tinted skin that the hue is not due to jaundice because the fæces are of a dark colour, one must make sure that the dark colour of the stools is not due to the presence either of blood or of black sulphurets, as then, and then only, will the conclusion be philosophically justifiable.

CHAPTER V.

TREATMENT OF HEPATIC DISEASES.

I HAVE now finished with what may be called the preliminary scientific part of my subject. But before entering upon the purely practical clinical portion of it, which will, of course, include not only the diagnosis and pathology, but also the treatment of every form of liver disease, I think it will be well for me, in order to save a great deal of what would otherwise in reality be mere therapeutical repetition, to make a few general remarks on the action and modes of employing what are usually spoken of as 'hepatic remedies,' in the same way as I have already for a similar reason briefly summarised the more important ordinary signs and symptoms of liver disease. For by thus, at once and for all, laying down the general therapeutical principles upon which their employment is founded, I shall not only be enabled to save a considerable amount of space, but at the same time accomplish what is equally important, namely a considerable economy of the reader's valuable time.

When speaking of the modes of applying the remedies, unless when otherwise specified, I shall consider my remarks to be applicable to every hepatic morbid state, no matter whether it be mere local action or general constitutional disturbance. For every change in bodily function is the direct product of some form or another of tissue alteration. To treat hepatic disease rationally, therefore, not only is it requisite that a correct diagnosis be first arrived at—as different forms of hepatic affections of course necessitate different kinds of treatment—but differently constituted and aged patients suffering from similar forms of liver disease not only require in many cases entirely different systems of treatment, but the same patients at different times, in different stages of the same affection, may even require what might be considered at the first glance to be diametrically opposed forms of therapeutical remedies. The matter before us is consequently not only wide, but difficult, and in order to discuss the subject of the therapeutics of hepatic diseases logically as well as practically, I must be allowed not alone considerable license in the selection of my materials, as well as of my data ; but the privilege of having their therapeutics regarded, not in fragments, but as one great and indivisible whole. For thus only can the individual value and therapeutical importance of hepatic remedies be thoroughly understood. This is the main reason (coupled with the

object already alluded to of economising the reader's time and my own space) why, instead of merely contenting myself with alluding to the special remedies applicable to particular forms of disease in their respective chapters, I have deemed it necessary to bestow a whole separate chapter to their joint consideration. Believing, as I do, that when once its contents have been mentally digested, and the general principles inculcated impressed upon the mind, the question of appropriate remedies in the special forms of hepatic derangements will be comparatively easily answered. For once the rationale of their therapeutic action is understood, there can exist but very little difficulty in comprehending the reasons for applying the different kinds recommended for employment under the various headings of treatment given at the end of each chapter on special classes of disease. I shall only require to mention their names, or at least do very little more than mention their names and doses, when alluding to the propriety of administering them in the treatment of the special case happening at the time to be the one immediately under consideration.

Fortunately for the sake of poor suffering humanity, the day of trusting to the Healing Powers of Nature in the treatment of disease is rapidly drawing to a close, and will soon become, it is to be hoped, as permanently moribund as the idea of

trusting to Nature to set efficiently a broken thigh-bone, or correctly reduce by herself a dislocated shoulder-joint. In such cases we know that the 'healing powers of Nature' invariably make a botch of the case. For while in the one instance she leaves to the patient, as a permanent souvenir of her handiwork, a crooked, in the other she as invariably bequeaths to him a shortened limb as an inheritance. The thoroughly enlightened practitioner of the present day has now learned from experience, if from nothing else, that contrary to the medical teaching which was so prominent a quarter of a century ago, Nature when left to herself makes nearly as unsatisfactory a physician as she is a bungling surgeon. That 'expectant medicine' is about as philosophic in its principles and satisfactory in its practice as would be the adoption of an 'expectant form of alimentation.' As God only helps those who help themselves, I consider expectant medicine not only wrong in theory, but pernicious in practice. And this may be said to be in no single instance more true than in the treatment of the class of diseases now under consideration.

Not only is 'expectant medicine' totally inadmissible in the treatment of hepatic affections, but, contrary to what many imagine, even 'routine practice' is equally reprehensible. In many instances being actually attended with danger. This in a great

measure arises from the as yet imperfectly appreciated circumstances that :—

1st. Scarcely any cases of liver disease—no matter whether they be cases of hydatids or hepatitis, cancer or gall-stones—ever run an identical course.

2nd. There is not a single form of liver disease—unless it be hepatitis—which, like a small-pox or a fever, will wear out, and cure itself.

3rd. Cases of liver disease, which at first sight appear to possess a perfectly identical pathology and parallel symptoms, and consequently to require similar treatments, are in the majority of instances, on closer inspection, found to pursue entirely different courses, and require different kinds of treatment. One cannot feel surprised at this, seeing what a complex organ the liver is, even when physiologically considered. For, as shown on page 58, it has no less than four entirely distinct normal functions to perform, one and all of which are liable to get out of order at different times and in different ways.

The reader can scarcely expect me, then, at the present moment and in the present place, to lay down special rules for the treatment of special cases. Indeed all that I shall now either pretend or attempt to do will be to lay before him the ordinary therapeutical knowledge we possess of the so-called hepatic remedies, adding a few hints which, from

personal experience, I believe, may be turned to useful account at the bedside. The success of their application will, however, of course greatly depend on the diagnostic skill in the first instance, and the innate therapeutical acumen in the second, of the prescriber. For

> Though Learning guides us healing herbs to pick,
> 'Tis Wisdom only makes them cure the sick.

Before entering upon the consideration of the employment of individual remedies in the treatment of hepatic diseases, I ought most emphatically to warn the young practitioner against :—

1st. Falling into the error—frequently committed—of imagining that patients labouring under diseases of the liver have a greater tolerance of remedies than those afflicted with more directly exhausting forms of disease : an idea which has, I believe, occasionally led to untoward consequences.

2nd. I would further call his attention to another important point, namely, that in the treatment of no other forms of disease is it of greater importance than in those the result of liver derangements to remember that certain therapeutical agents have a special action not only on one, but on two or more organs or tissues of the body at the same time. Otherwise, while doing good in one way, he may be actually doing injury in another. The twofold action of certain drugs is well illustrated, for example, in belladonna, which

acts specifically both on the pupil and larynx ; in iodide of potassium, which acts equally specifically both on the kidneys and salivary glands; while mercury, the most common of all hepatic remedies, at one and the same time has a special and triple action of its own on three entirely different organs—to wit, the liver, the intestinal canal, and the salivary glands.

3rd. Let it never be forgotten that all remedies are also poisons, the amount administered alone constituting their right to be designated as the one or as the other.

4th. The treatment of liver diseases naturally divides itself into a general and a special. In the consideration of the former, constitutional symptoms more than local morbid effects ought to occupy chief attention. In the consideration of the latter, the seats or localities of the pathogenic action require special attention. Consequently, in order to be able to treat a patient labouring under hepatic disease rationally, it is quite as necessary for one to have a clear notion of the etiology and natural career of the affection under which his patient labours, as to possess a knowledge of its signs, symptoms, and modes of termination.

Mercurials.

The first remedy to which I must call special attention is the well-known old-fashioned mercurial, which, after having been in general use among all ranks and classes of society for generations, may be still looked upon as the physician's mainstay in the treatment of the majority of liver cases. Every housewife knows that a dose of calomel at bed-time, followed by a black draught in the morning, will suffice, in the vast majority of cases, to cure an attack of biliousness, and that too within twenty-four hours after its administration. At the present moment, however, a change has come over the spirit of the physician's mercurial dream, and the poor old drug has been placed at the bar like a suspected criminal 'on trial,' on account of experimental physiologists having found, that when administered to the canine species, it does not behave itself in what, according to old-fashioned notions, might be called an orthodox manner. As some of my readers may expect me not to pass over in silence the points in dispute, I shall, as briefly and concisely as I can, give my views of the special action of mercury on the human liver.

There was a time when mercury was administered in all cases of liver diseases quite irrespective of their cause. Now, however, although men are fortunately

becoming more careful in the employment of the drug, it is still often misapplied from there being many mistaken notions regarding its therapeutical action. It was at one time thought that mercurials stimulated the liver to secrete bile. But since experimental physiology has shown that they possess no such action on dogs, many have gone to the opposite extreme, and declared that as mercurials do not stimulate the liver to secrete bile in dogs, their benefit in human hepatic disease has been a delusion; and the dark stools following upon their employment but the result of the sulphuret of mercury formed in the intestines.

The hostile therapeutists stand opposed to each other thus: Those of the practical clinical school declare that mercury is a powerful hepatic biliary stimulant, while those who adopt the views of the experimental physiological school as emphatically declare that mercury has no effect whatever in exciting or in increasing the biliary secretion, either in men or in dogs. Now comes the question which side is right and which side is wrong? Both sides most assuredly cannot be right, though both sides equally certainly may be wrong!

To reconcile not only these two different views, but even the facts upon which each of them is based, on scientific principles, has hitherto been deemed impossible, except upon the supposition that the con-

stitution of the dog, as regards the action of mercury upon the liver, is entirely different from that of man.

Mercury, it was said, might be a powerful hepatic biliary stimulant in the human, and yet perfectly inert in the canine species. This opinion was arrived at by a process of reasoning from analogy. For it is not only a well-known, but a perfectly incontrovertible fact that not only one, but many therapeutic and toxic substances act not alone with varying degrees of intensity, but even in a diametrically contrary manner, when administered in precisely the same form and in the same way to different species of animals. I could easily cite a dozen of examples of the actions of different poisons in proof of this statement; but it is quite unnecessary for me to do more than remind the reader of the well-known and most extraordinary one, that goats eat hemlock with impunity, while sheep instantly succumb to its poisonous action. Nay more, that the milk of the goat fed upon hemlock leaves poisons the adult human being, while the little delicate kid not only relishes, but actually thrives upon, its mother's poisoned milk. It is perfectly evident, then, that the contradictory effects of poisonous substances when administered to different species of animals, may, with an apparent good show of reason, be given as the true explanation of the contradictory results obtained from the action of mercurials on human and canine livers.

This explanation does not at all satisfy me, for I have yet to be convinced that mercury *does* act differently upon dogs from what it does upon men. My experiments upon the toxic effects of mercury both in suddenly administered large doses, and with insidiously daily administered small doses, varying in duration of time from fourteen to one hundred and twenty days, have led me to the belief that the action of mercury on the liver of the dog is precisely the same as it is upon the liver of the human being. For be the rationale of the action of mercury upon the hepatic organism what it may, I hold it as an undeniable fact, that after the sudden administration of a large dose of mercury to healthy dogs as well as to healthy men, a variable but always considerable increase of bile is detectible in the fæces both by the pigmentary and bile-acid tests.

My belief is, that at least one-half, if not three-quarters, of the cause of the dispute regarding the action of mercury on the biliary function arises from the slipshod manner in which the writers engaged in the discussion employ the words 'secretion' and 'excretion,' often, it appears to me, using the one term, when it is perfectly evident, to the reflecting reader, that they ought to employ the other.

I do not imagine that there is a single person who has taken part in the discussion that will seek to deny that, after a smart dose of mercury, not only

do the human fæces look as if they were loaded with bile ; but that the patient even occasionally complains that the passage of the stool through the anal orifice has produced a feeling of smarting or hot scalding, which smarting can be due to nothing else than the irritation produced by an excess of the bile-acids in the stool. Moreover, I think no one at all versed in the literature of liver diseases will seek to deny that several independent observers are said to have noticed that an increased flow of bile has taken place from accidental human biliary fistulæ after the administration of a brisk mercurial cathartic to the patient. Every one, I believe, will, however, at the same time admit that neither an increased elimination of bile by the stool, nor through the fistulous opening directly connecting the gall-bladder with the exterior of the body, is any proof whatever of an increased SEcretion of bile by the liver having taken place ; but is merely positive proof that an increased EXcretion of bile has occurred, and that in either case the expelled bile may not have issued, and most probably did not issue directly from the liver at all, but only from the gall-bladder, which had received it from the liver some time previously, and had it stored up in its interior ready for excretion at any given moment.

Not only may bile be secreted in great quantity, and yet not excreted in consequence of the gall-

bladder—its reservoir—being sufficiently capacious to retain it; but a large quantity of bile may be excreted at a time when little or none at all is being secreted; that excreted being merely the bile that had been secreted some time previously, and been retained stored up in the gall-bladder, as above said, until the proper moment for its excretion arrived. While again, according to my views of the matter, this excretion of the pent-up bile independent of secretion may be entirely due to the brisk action of a mercurial—in the following wise :—

Bile is only expelled from the gall-bladder as a result of the mechanical effect of its contraction. Its muscular contraction is called into play by reflex nervous action. And, in the normal state at least, this is brought about by the periodic stimulus given to the peristaltic action of the duodenum during the passage through it of the irritating acid chyme—from the stomach. The stimulating effects of the acid chyme on the muscular coat of the intestines being communicated by reflex action, back along the common bile-duct from its duodenal orifice, to the muscular coat of the gall-bladder, which in its turn is thereby excited to contraction, and expels the necessary amount of bile into the intestines to play its physiological chemical *rôle* in the digestive process; by which mechanical contraction of the gall-bladder, moreover, its contents—bile—if not expelled, under

the normal circumstances, along the common bile-duct into the duodenum, may be under the abnormal circumstances expelled through a fistulous opening in the abdominal walls directly to the exterior of the body, and give rise to the condition that has been described as above by different observers. In opposition to the latter part of this theory, again, we have the, at first sight, apparently irreconcilable statement of experimental physiologists that mercurials have *no effect whatever* on the quantity of bile eliminated through a biliary fistula in a dog. How is this? To me it appears to be not another example of scientific discord, but simply of scientific 'harmony not understood.' And now for the explanation. What is it that the experimental physiologists tell us? Merely this—that when a dose of calomel is given to a dog with a gall-bladder fistula, after the common bile-duct has been secured by a ligature, in order to prevent any bile escaping unnoticed into the intestines, *no visible* increase takes place in the quantity of bile flowing from the orifice of the fistula. Now, this is of course a perfectly conclusive statement. But of what? Certainly not that the administration of a dose of calomel does not produce bilious stools, either in a dog or in a man, but merely that a dose of calomel does not increase the *se*cretion of bile by the liver. Which is quite another thing. The emptying of a distended gall-bladder of its bile being a thing

which a dose of calomel *can do*. The stimulating of a healthy liver to *se*crete bile being a thing which a dose of calomel *cannot* do.

Some clear-headed reader may feel inclined to put to me the question, 'But how, then, do you reconcile the negative statement of the physiologists with the positive one of the practical physicians who say that a dose of calomel increases the flow of bile from the fistulous opening of their patient's gall-bladder?' Easily enough. An experimental physiologist's biliary fistula is in most instances a very different thing in its anatomical relations from the vast majority of biliary fistulæ which originate as the result of disease in the human being. As I well know from practical experience, having made many of them, the fistula in the dog generally opens directly into the free top end of the gall-bladder, and the gall-bladder, not having any longer its natural contents allowed to accumulate in it, very soon dwindles (in function at least) into a mere bile tube. So that it ceases to be any longer a reservoir of bile, from which a dose of calomel, by causing it to contract, can suddenly expel its accumulated contents, and that too for the simplest reason of all, namely that it contains nothing to expel. In the human being, on the other hand, the fistulous orifice in the gall-bladder is much more often in its side than anywhere else, and in the cul-de-sac formed by the opposite

side of the gall-bladder, together with the pendulous globular end of the viscus, bile may not alone daily and hourly collect, but most probably does accumulate there in considerable quantity, so that when a dose of calomel is given to this sort of biliary-fistulous patient, the dilapidated organ contracts and suddenly expels the collected bile it chances to contain in its interior at the time, just as a healthy gall-bladder would do under similar circumstances. The bile that comes away through the fistulous opening in the dilapidated gall-bladder not being *se*creted at the time of its expulsion, but merely *ex*creted from its reservoir by the stimulating effects communicated to the gall-bladder from the duodenum excited into brisk peristaltic action by the mercurial purgative administered to the patient.

Perhaps not more than one patient in ten may have this peculiar form of gall-bladder fistula (associated with a cul-de-sac admitting of the accumulation of bile in its interior), and consequently the effects of a dose of calomel on the other nine would be followed by the same negative results as in the dog with the artificial biliary fistula. This is the only way in which I can reconcile the discordant statements of men on either side, whose experimental powers, as well as veracity, are unimpeachable, and I think it is not alone the most generous, but at the same time the most philosophic way of meeting the difficulty.

Having said this much regarding the dispute, I may now venture to give my ideas regarding the mode of the immediate action of mercury when it is given in a purgative dose to a bilious patient.

As every medical practitioner well knows, when he administers a sufficiently large dose of a cathartic mercurial to a bilious individual, a large, black, tarry stool comes away.

Look again at the effect of calomel on the stools of a child at the breast. Normally the stools are of a pale straw colour; but give a dose of a mercurial, and immediately they become of a distinctly bilious green hue. The bile being in many instances in sufficient quantity and sufficiently concentrated to scald the anus during its exit. It is not, however, the bile which has just been *se*creted that then alone comes away, but the accumulation of thickened tarry bile, which has been, perhaps for days or weeks, stored up in the gall-bladder, that the mercurial has all of a sudden expelled from the viscus. The sudden expulsion of the accumulated bile from the gall-bladder being due to the stimulating effect of the mercurial on the peristaltic action of the duodenum. Its irritative or, physiologically speaking, stimulative effects on which being communicated, by reflex nervous action, along the bile-duct to the gall-bladder, and thereby exciting to immediate contraction its muscular coat. By which contraction the biliary contents

of the viscus are suddenly expelled into the intestines, and give origin to the tarry bilious stools.

While giving this as my theory of the immediate effects of mercury on a bilious patient, it is by no means all that I have to say upon the rationale of the curative effects of mercury in hepatic diseases. An equally important and more intricate one has now to be considered, namely, its beneficial action in all the various forms of congestion of the liver, and consequently, of course, in all cases of jaundice the result of hepatic congestion.

While admitting that there is sufficient evidence derivable from physiological sources to prove that mercury has no power to stimulate the normal liver to secrete bile, I shall now endeavour to prove that I am justified in holding and in promulgating the theory that in certain cases of diseased liver, where the biliary secretion is retarded, or even arrested, in consequence of a congested condition of the tissues of the liver, mercury has a powerful, though only an indirect, effect in restoring the biliary secretion. Not alone in the human, but equally so in the canine, bovine, and equine species. And this it does, I believe, by means of its antiphlogistic action upon the hepatic capillaries; by subduing, if not indirectly actually removing, the congested condition of the blood-vessels, it relieves the secreting structures from the mechanical pressure arising from the congestion

of the blood-vessels, which prevents the hepatic cells from secreting bile.

If this view of the action of mercurials, when administered in cases of congestion of the liver, be equally tenable with the theory of the immediate power they have in mechanically empyting an overloaded gall-bladder in a bilious patient (coupled also, perhaps, with their antiphlogistic action on the congested condition of the liver with which an attack of biliousness is usually associated), we have at least advanced two steps in the right direction towards arriving at correct notions regarding the action of mercurials in the treatment of liver diseases, and I think are thereby prepared to go yet another step, and attempt the solution of the problem of the beneficial action of mercury in the various other forms of liver disease beyond those merely included in the generic terms of biliousness and congested liver.

The further beneficial effect of mercurials, in many other forms of hepatic derangement, appears to me to consist in a great measure in the powerful effect repeated doses—even small doses—of mercury have upon the blood, particularly on its red corpuscles. A large dose of mercury, by inducing liquid stools, not only reduces to a certain extent the total volume of blood in the circulation, but it at the same time impoverishes the blood by its disintegrating power on the cell-walls of the red

corpuscles, and thereby allowing their nutritive contents to escape. Small doses again—not large enough to produce purging—though they may not directly reduce the total volume of the blood, still nevertheless act by impoverishing it. For no matter however small a quantity of mercury finds its way into the circulation, I believe from the results furnished to me by my experiments on the action of mercury on animals, that a directly proportional impoverishment of the blood invariably takes place. Sir Thomas Watson has poetically said that mercury can blanch the cheek of the rose to the whiteness of the lily, and nothing, I believe, is more true. For in experimenting on animals, I have found the prolonged use of mercury reduce the red blood-corpuscles in a marked manner. Reckoning by the eye when they are viewed through the microscope, I should be inclined to say, at least one-fourth. From this it is easy to understand how mercury acts in inflammatory affections; and as in the majority of cases of jaundice from suppression, the stoppage of the biliary secretion is due to active congestion of the liver, mercury proves beneficial in such cases, not by directly stimulating the suppressed biliary secretion, but by simply removing the obstacle to its re-establishment; namely, the hepatic congestion in the two ways just indicated.

As a good illustration of the correctness of

this theory regarding the action of mercurials in cases of jaundice arising from hepatic congestion, I shall quote the following case, which appeared in the Hospital Reports of the 'Lancet' of the 7th December, 1861. The case is headed, 'Intense Congestion of the Liver, simulating an Abdominal Tumour:'—

Alex. E——, aged forty-eight, was admitted into St. Bartholomew's Hospital, under the care of Dr. Farre, on the 17th October, 1861. The patient had, it appeared, been suffering from jaundice during six weeks. He stated that the tumour in the epigastrium began at the same time as the yellowness of the skin. On examination, a prominent swelling was noticed in the epigastric region, possessing an indistinct feeling of fluctuation, but it was found to be continuous with the liver. The motions were not bilious, but were of a pipeclay colour, and the urine looked like pure bile. Three grains of blue pill and two of Barbadoes aloes were ordered every night. By the 25th the hepatic tumour was less, and the icterus was disappearing. On November 4th the urine was clearer and full of lithates. The conjunctivæ were now the only parts observed to be of a yellow colour.

November 11.—Although the pills had been continued up to this date, the mouth was not sore. The urine and stools were natural, and the patient

was convalescent. A few days afterwards he left the hospital. The result, as the reporter stated, clearly proved not only that the swelling was from a highly congested liver, but also that the jaundice depended on this state.

In this case it is evident that the primary beneficial action of the mercury was to reduce the congested state of the hepatic organ, and no one, I think, would venture to say that this was accomplished by any power that the mercury possessed of exciting by means of stimulation the liver to secrete bile. If, then, the above views of the therapeutical action of mercurials be correct, it is easy to understand how, while in cases of jaundice from hepatic congestion it is beneficial, in those again from a permanent obstruction of the common bile-duct in any part of its course, the administration of mercury or any other lowering medicine must prove detrimental by hastening the fatal termination.

Like most other men actively engaged in practice, I have three favourite grades of mercurials, of gradually decreasing strength, suitable for patients of different ages, sexes, and constitutions.

At the head of the list stands our old and venerable friend Calomel, in his from three to six grain doses. Next in order of sequence comes Blue Pill, which again in its turn is followed by the less severely acting Grey Powder. One and all of these to

be given at bed-time; but *not to be followed in the morning by a purgative*—as was the almost habitual practice some years ago—unless the bowels will not act within twelve hours without one. Severe purgation I have over and over again found to be not only uncalled for, but even detrimental, in all except fat, fleshy, plethoric patients, who appear to require reducing. All that is required of the mercurial—unless we desire to salivate—being to cause one free and copious action of the bowels. Not half-a-dozen, as was formerly considered to be requisite. Consequently, before telling a patient to take opening medicine in the morning after a nocturnal dose of mercurial, I always enquire if the bowels are easily moved, and unless they are not I prescribe none—except he be at the same time a person of the above-described constitutional type. Should I consider a matinal purgative desirable, then I usually select the one the patient is most accustomed to, regulating its strength according to circumstances, but in all cases giving strict injunctions not to take the purgative along with the mercurial at night. For I have the idea that no matter in what form the mercury be given, it always acts best upon the biliary function of the liver, through its direct action upon the blood, when administered alone. If, however, prescribing for a trifling bilious attack, I pay no attention to this rule, and may advise a five-grain

pill of equal parts of ext. colocynth and blue pill to be taken at bed-time; but whenever I desire to act on the biliary function of the liver thoroughly, I give the mercurial alone, following it up with the purgative, when necessary, eight or ten hours later, with the view of simply increasing the peristaltic action of the duodenum and by reflex action stimulating the gall-bladder to contract more powerfully and the better be able to expel its bilious contents. Moreover, for a precisely similar reason—namely, non-interference with the cholagogic action of the mercury—it is that I prefer giving it on an empty stomach. For if the stomach is loaded with food when the drug is taken, or if food is introduced into the stomach after the mercury has been administered, and before it has had time to produce its therapeutical action through the blood on the liver, not one half of its beneficial effects are, I believe, obtained.

Alkalies in Hepatic Disease.

Although mercury has not, there are some substances which have, the power of directly exciting the flow of bile. Just as there are substances which directly excite the flow of saliva. Among these the mineral acids and soluble alkalies hold the first rank. It may seem strange that acids and alkalies should be here placed in juxtaposition; but the reason of this arrangement will immediately appear.

According to a physiological law, acid substances have the power of exciting alkaline secretions, and alkaline substances of stimulating acid secretions.

Bile being an alkaline secretion, we can therefore have no difficulty in understanding how the mineral acids act in cases of jaundice from suppression induced by enervation. They simply stimulate the secretion of bile. Bernard proved this by direct experiment, for he found that acetic acid applied to the duodenal orifice of the bile-duct caused an instantaneous expulsion of bile from the gall-bladder.

It is not so easy, however, to comprehend the action of alkalies in similar cases.. My explanation of their action is as follows :—When taken after food on a full, and when taken before food on an empty, stomach, the action of an alkali is entirely different. After food, and during digestion, the stomach contains a quantity of acid gastric juice, and an alkali taken then only neutralises the acid of the gastric juice. On the other hand, when an alkaline substance is introduced into an empty stomach, it acts according to the general law of exciting an acid secretion ; and consequently an immediate flow of acid gastric juice takes place. And I believe it is the excess of this acid gastric juice, which, on reaching the duodenum, not only stimulates the secretion of the bile by the hepatic cells, but also excites the excretion from the gall-

bladder of the alkaline bile. Just as the mineral acids do under similar circumstances. One remark further is, however, necessary. The quantity of alkali employed for the purpose of stimulating the secretion, or of exciting the excretion of the already secreted bile, must be small, for if much be used, the greater part of the acid of the gastric juice will be rendered useless, in consequence of its being neutralised by the alkali as fast as it is secreted. Moreover it may be laid down as a general rule, that when we desire to increase the flow of bile by means of a mineral acid, the acid must be given *after food*. When, on the other hand, an alkali is selected for that purpose, the alkali must be administered *before food*.

For obvious reasons, both alkalies and acids are counter-indicated in cases of jaundice resulting from active congestion of the liver; and it is equally evident that they can be of no direct service in jaundice arising from occlusion of the bile-duct, where our object should rather be to diminish than to increase the secretion of bile.

I believe that all the contradictory results which have been recorded by experimentalists, regarding the effects of carbonate of soda on the excretion of bile by dogs, are entirely due to sufficient attention not having been paid to these important physiological facts. For example, Dr. Fraser, in an article

in the 'Edinburgh Medical Journal' for April 1871, tells us that large doses of carbonate of soda decreased both the *fluids* and solids of the bile, when administered to dogs with biliary fistulæ. And no wonder that they did. For had he given the animals a large dose of kitchen salt, I have little doubt he would have found a precisely similar result, and that, too, for a precisely similar reason. Namely, that the salt would diminish the quantity of liquid in the blood-vessels—from a palpable cause, as would be manifested by the thirst it would create—and there would be less for the hepatic secreting cells to obtain for their requirements, and hence a diminution in the amount of bile issuing from the orifices of the biliary fistulæ.

It is not with either Dr. Fraser's, Nassi's, or Rohrig's facts 'on this supposed action of carbonate of soda on the biliary secretion of dogs' that I find fault, but it is with the modes of theorising adopted by one and all of these gentlemen that I disagree. Professor Rutherford's results are even opposed to theirs; for he found that carbonate of soda in similar cases slightly increased the biliary secretion ('British Medical Journal,' 1879, p. 105).

Alkalies, or at least some alkalies, possess certain other properties besides those to which allusion has just been made, which may be usefully turned to account in the treatment of jaundice from gall-stone

obstructions. To this property of alkalies I shall specially allude under the head of the treatment of gall-stones.

Chloride of Ammonium.

To the peculiar action of chloride of ammonium I must here direct special attention, as by some of our best writers on the tropical forms of hepatic disease it is said to be quite equal to mercury in its beneficial effects, without possessing any of the deleterious qualities of the mineral. Chloride of ammonium is most suitable in cases of hepatic congestion, which it speedily relieves when administered in full doses—from twenty to thirty grains—three or four times a day. The pain rapidly disappears, and the size of the liver diminishes within forty-eight hours after beginning the administration of the remedy. As far as I am aware, we are indebted to Dr. William Stewart for first having called the attention of home practitioners to the valuable therapeutic properties of the chloride of ammonium in hepatic congestions. Following his advice, I very frequently prescribe it in the doses he recommended, as above given, and with, in general, marked beneficial results. It often speedily relieves the pain, stimulates the appetite, and increases the flow of urine; but how it acts in this way on the liver, in reducing the congested state of the organ (which of course is the

cause of the pain), I know not, for in no sense of the word can it be called an antiphlogistic. On the contrary, it seems rather to be a stimulant of both the nervous and circulating systems, for which reason I suppose it is that Dr. Stewart considers that a dry and hot skin counter-indicates its administration.

Vegetable Remedies in Hepatic Disease.

To persons much in the habit of prescribing for liver affections it is well known that a great number of new hepatic remedies have been recently introduced into Europe from America—to wit, podophyllin, and a number of other vegetable drugs which I shall presently allude to. But I must remind my readers that even before this we had no lack of either vegetable or mineral substances in our pharmacopœia of considerable repute in precisely the same class of affections, among the most noted of which I may mention taraxacum, aloes, colocynth, croton oil, rhubarb, colchicum, scammony, senna, ipecacuanha, and jalap. At the same time it literally abounds in mineral ones, besides mere mercurials, among the best known and most valued of which are the sulphates of soda and magnesia, the bicarbonates of soda and potash. While, again, among the semi-organic and semi-inorganic may be named the chlo-

ride of ammonium, the benzoates of soda and potash, and the salicylate of soda. One and all of which remedies are more or less powerful stimulants of the biliary secretion. The action of some of them, however, is not only peculiar, but special. I shall now therefore speak of their therapeutical actions somewhat in detail, and, to begin with, I shall direct attention to the vegetable drug which is thought by many to possess equally beneficial curative powers with mercury, especially in all cases of torpid liver, and of jaundice the result of a suppression of the biliary secretion. I allude to podophyllin, or, as it is vernacularly called by our American cousins, May-apple.

This remedy was first introduced to the profession nearly a quarter of a century ago, by an American physician who believed it to possess, as he said, both the alterative and purgative properties of mercury. As an alterative, he recommended the resin to be given in doses varying from one-eighth to one-fourth of a grain, three times a day; as a purgative, from one-fourth to one grain, as a single dose. I have given this remedy a tolerably fair trial, and although it seems to me to be very useful as a purgative in hepatic disease, and to increase the flow of bile, I have found it open to two objections: firstly, its action is not always certain; and, secondly, in delicate females it gives rise to a good deal of griping.

This latter objection can, however, to a certain extent, be counteracted, by combining the remedy with hyoscyamus. On the whole, I prefer mercurials to podophyllin resin, and only administer the latter in slight cases of jaundice, or in those where mercurials are counter-indicated.

For example, in cases of feeble liver, where there is an insufficient secretion of bile from want of nervous power, podophyllin is decidedly of service, for in such cases mercury is of course counter-indicated. Moreover, podophyllin can be advantageously combined with vegetable tonics, and, when given along with gentian or strychnine, forms an admirable hepatic stimulant in some of the cases usually denominated 'torpid liver.'

Dr. Dobell says that the best way by far of administering podophyllin is to dissolve it in spirits of wine in the proportion of gr. j. to the ounce, and combine it with essence of ginger in the proportion of ʒjss. to an ounce; a teaspoonful of this given in a wineglassful of water every night, or every second or third night, will secure all the advantages of podophyllin without any chance of incurring those disadvantages which so often result when it is given in pills ('British Medical Journal,' May 24, 1879).

I cannot refrain from here making a few remarks on what I consider the injudicious employment of podophyllin, notwithstanding that I shall have again

to refer to it when on the subject of the treatment of gall-stones. Like every new remedy, podophyllin and its eleven sister American new hepatic stimulants have to run the risk of falling into disfavour in consequence of their too ardent admirers blindly prescribing them in all cases of hepatic disease, in many of which they must of necessity prove unsuitable, if not even detrimental. In cases of jaundice, for example, they must of necessity, at one and the same time, be the bane and the antidote. The bane in *all* cases of jaundice from obstruction. The antidote only in some cases of jaundice from suppression.

In all hepatic diseases, independent of a diminution in the secretion of bile, not alone one, but all hepatic stimulants do absolutely no good, and even sometimes do actual harm by adding to the mischief already accruing from an excess in the system of secreted and unused bile. So long as the bile they excite can be poured into the intestines—even should they excite too great a secretion—they can do no harm, but so soon as they excite a secretion of bile which cannot find its way into the intestines, in consequence of some cause of occlusion or other existing in the common duct, then one and every hepatic stimulant does harm ; for every particle of bile secreted, and not excreted, but adds to the stock pent up in the gall-bladder and hepatic ducts, which gradually becomes more and more concentrated in its

consistence, from the fact that there is a constant absorption of the aqueous particles of the bile, by means of a process of capillary osmosis, going on during the whole time it is shut up in its reservoir and ducts. When the liver during the intervals of digestion secretes merely sufficient bile to meet the requirements of the succeeding meal, by the end of the digestive process the gall-bladder has entirely emptied itself, and is again quite ready to receive a fresh supply. Whereas, when the liver secretes more bile than it can get rid of, the excess remains in the gall-bladder and ducts, and by its presence there leads, sooner or later, to the entire disorganisation of the secreting tissue of the liver, in consequence of the deleterious backward pressure exerted by the pent-up secretion on the hepatic cells.

Taraxacum has been widely used in hepatic disease associated with jaundice, and is, I think justly, believed to be particularly well adapted to the treatment of cases arising from congestion. As in such cases I generally combine it with more potent drugs, my experience with this remedy when administered by itself has been too limited to admit of my offering an opinion of its value in an uncombined state. I may, however, with perfect propriety, I think, speak most favourably of it when combined along with an alkali, such as soda or potash. My favourite prescription when I use taraxacum is

R Succi taraxaci	. .	ʒxvj.
Sodæ bicarb.	. . .	ʒiij.
Sodæ sulphatis	. .	ʒvj.
Inf. calumbæ ad	. .	℥vj.

M.

Sig.: Shake well, and take a tablespoonful in six ounces of water three times a day.

Dr. Washington again, who practises in Georgia, thinks that Old-man's-beard (*Chionanthus virginica*) should be placed in the foremost rank as a curative agent in all the various forms of liver disease incidental to malarial poisoning. According to him it not only stimulates the liver to secrete bile, but improves the digestive and assimilative functions, and thereby acts as a general tonic, and proves exceedingly useful in the dropsical concomitants of malarial jaundice, either in their acute or in their chronic forms. His mode of administering it is to give a teaspoonful of a strong aqueous extract three or four times a day; and he says this will succeed in some of the more obstinate forms of intermittent malarial fever poisoning, when quinine has failed.

In addition to these vegetable hepatic stimulants, the profession has recently been strongly invited by advertising druggists to patronise other six newly proposed remedies for liver diseases, of varying degrees of activity, all said to be of undoubted value when given in the following doses :—

Baptisin (wild indigo), gr. ij., gr. iv. ; Euonymin (wahoo), gr. j., gr. ij. ; Iridin (blue flag), gr. ij. ; Juglandin (butternut), gr. ij. ; Phytolaccin (poke-root), gr. $\frac{1}{4}$, gr. j. ; and Leptandrin, gr. ij.

One and all of these preparations are kept, made up in the foregoing mentioned doses, by druggists, in the elegant form of pearl-coated pills.

Some of the fore-mentioned so-called hepatic stimulants possess a double salutary action in cases of biliousness, in not only stimulating the secretion, but also the excretion of bile by the intestines ; while others again limit their beneficial effects entirely to stimulating the secreting hepatic cells. Thus, while taraxacum, ipecacuanha, colchicum, leptandrin, and benzoate of soda merely act upon the liver, aloes, colocynth, calomel, podophyllin, jalap, salicylate of soda, sulphate of potash, and sulphate of soda not only stimulate the secretion of bile, but by their purgative action excite its expulsion from the intestines, and thus exert a double beneficial influence on bilious patients.

Germicides.

The new word ' Germicides ' may perhaps to some seem peculiar, especially in connection with diseases of the liver. But it will appear nothing extraordinary to the reflecting medical man who has been keeping himself *au courant* with the discoveries that

have recently been made regarding the important part played by disease-germs in all hepatic affections of a malarial and epidemic character. For in the mighty revolution which the study of germ pathogenic action is rapidly producing in our ideas of the etiology and pathology of all kinds of epidemics, as well as of many forms of sporadic jaundice, he must have perceived it is but the forerunner of an equally great change taking place in our systems of treatment.

Here, as everywhere else, it may be said that rational medicine brooks neither curb nor restraint. Her movements always are, as they ought to be, quite as untrammelled as those of the course of a bird in the air or a fish in the sea. And now that the marvellous success of antiseptic surgery has given a clue to some at least of the lines along which she may advantageously proceed, it is impossible to predict how soon or how effectively the medical therapeutics of liver disease will profit by the example set by her twin sister surgery.

The end and the aim of therapeutics is, and ought to be, the discovery of specifics. Specifics, not in the narrow sense of mere panaceas, but in the broad one of true curative agents. And most fortunately for us, of all the various forms of human disease set forth in our compendious nosology, it is those which have already been, and are still about to be, philo-

sophically enrolled under the heading of 'Parasitical Affections'—in which category of course all germ diseases must logically come—which offer us the fairest chance for the discovery of their specifics.

Already we possess a number of specifics more or less infallible, and nearly every one of them is a parasiticide. I am now speaking solely of internal remedies, leaving altogether aside the numerous external applications with which are successfully destroyed the many vegetable and animal parasites affecting the skin and its appendages.

Limiting myself to the consideration of those alone which make the internal tissues, cavities, and organs of the body their habitats, I may tabulate our at present known more or less infallible specifics as :—

Oil of male fern in tapeworm.
Quinine in ague.
Acid perchloride of iron in erysipelas.
Santonine in ascarides.
Mercury in syphilis.
Salicylic acid in sibbens.
Chaulmoogra oil in leprosy.
Salicylate of quinine in typhoid.
Cod-liver oil in scrofula.
Belladonna in asthma.
Conia in nerve-spasm.
Salicylate of soda in rheumatic fever.
Opium in lead-colic.

One and all of these, though not infallible—for nothing in this world is infallible—when judiciously employed in appropriate cases, are, in the true sense of the word, specifics. That is to say, they possess a special and peculiarly curative power in the various forms of disease above mentioned, ameliorating the sufferings of all, and tending to prolong the lives of most of the patients labouring under them.

To this list, I anticipate, will soon be added many more, from a new era having dawned on therapeutical studies. For, while hitherto the action of remedies in the cure of disease has been entirely limited to the observation of their physiological effects on the healthy frames of the lower animals, or their therapeutical effects on the diseased constitution of man. Now that we have learned that many forms of human diseases are due to the action of the microscopic organisms included in the generic term disease-germs, an entirely new field of enquiry has opened out to us. All that we want is to discover agents which, while they prove fatal to germ existence, will be harmless to the germ's host to whom they are administered. Seeing, then, that the primary part in the therapeutical enquiry is simply to ascertain how disease-germs can be most readily killed, all the preliminary observations may be conducted in the chemical laboratory, or even in the study; all the implements that are requisite for the re-

search being a good microscope, a few flasks containing animal fluids or tissue infusions, and one or two simple chemical reagents. I have been thus working in my consulting room for months, and nobody has ever noticed anything particular going on, except when I called their attention to my work.

After these remarks it will, I think, surprise no one, if I add that the 'great secret' in the treatment of all hepatic germ-diseases is precisely the same as it is in that of the various kinds of what have hitherto been looked upon as specially parasitical forms of hepatic affections ; namely, the slaughter of the offending parasite. Kill or submit to be killed being seemingly an inevitable law of animal existence, for life appears to be but little else than one long fight between the destroyer and his victim. The wellbeing of one individual depending in a great measure upon his powers and his opportunities of killing and consuming another. Indeed, it may truthfully be said, that the maintenance of life, even in the vegetable, as well as in the animal world, consists in an uninterrupted succession of correlated consonance and disconsonance. For man destroys the sheep which consume the grass, that grew and developed out of the elements which constituted his ancestors' frames. It is not however always, as in this case, the stronger that destroys and consumes the weaker. In the group of diseases with which we

are at present dealing, it is actually the reverse. For it is tiny and apparently individually weak disease-germs which destroy and consume the stronger, the human being, their involuntary host. And it is a crusade against their lives I am now about to preach. For although to live and let live is a motto as philosophic in its conception as it is humane in its precept, self-preservation being the first law of nature, we, as the healers of disease, must put it entirely out of sight, and do our utmost to destroy disease-germs in every shape and character. For if they are let alone, they will most assuredly have no compunction in destroying us. It must no doubt be supposed that every living thing in nature was created some wise end to fill; but what the wise end in the creation of disease-germs can possibly have been, it completely beats me to discover. For while I fail to see that they do any thing or any creature any good, I am painfully conscious that a vast amount of bodily and mental human suffering must be laid to their charge. For they not only engender painful and exhausting diseases of a transient character, and sometimes give rise to what are called tertiary and quaternary signs and symptoms of disease which neither time nor treatment can remove, but often hurl the victims of their onslaught with scant warning into an unwished-for tomb.

There is one great and important fact that I

desire the reader to bear steadily in mind while perusing what I have to say regarding the treatment of hepatic germ diseases. Which is that infinitesimally minute though disease-germs be, they must be looked upon in the light of as true corporeal entities as we are ourselves. For germs, like men, are born, grow up to sexual maturity, perform for a definite and allotted time the functions for which they were created, decay, die, and disappear. And just as human life may last but a few brief moments, or continue a hundred or more years, so perhaps may the life of a disease-germ be as brief or as extended. While, by a similar process of reasoning from analogy, we may further philosophically conclude that while we have the power to curtail, though not to extend, the allotted span of human life, so in like manner we possess the power of artificially curtailing the existence of germs.

Our object being to discover the safest and the simplest means of killing and exterminating disease-germs from the tissues and fluids of the human body, and thereby mitigating, if we cannot entirely put a stop to, the effects of their ravages, in the shape of local lesions as well as constitutional effects. The salient points requiring attention are, how we can with the least detriment to their host counteract—

1. The development of colonies of germs in the tissues or vessels of the human body.

2. The blood fermentation induced by the general contamination and spread of germs throughout the system.

3. The fever resulting from the growth and development of the disease-germs.

4. The cerebral and other nerve symptoms arising from the circulation in the blood of toxic products engendered by the fermentation of the fluid and solid constituents of the body through germ growth and development.

5. The consequent exhaustion of the patient's vital powers.

It is easy enough to kill germs, but unfortunately not so easy to kill them quickly and effectually, without at the same time doing injury to their host. The reason of this is very simple. Disease-germs have a marvellous power of reducing human vitality. A few days, ay, even a few hours, will sometimes suffice for them to destroy life. It often happens in the course of hepatic germ diseases that death occurs quite suddenly and with scarcely any warning, making it appear as if a mere trifle were sometimes sufficient to totally extinguish the flickering flame of life. Which fact of itself makes it all the more necessary for the practitioner to exercise the greatest care in the selection of remedies in treating germ diseases. A drug which might at least be taken with perfect impunity, if not even with actual ad-

vantage, in some cases of jaundice, may be attended with fatal consequences if administered to a patient labouring under the exhausting effects of disease-germs. In fact I am not quite certain that some of the cases of supposed unaccountably sudden death might not with some show of reason be attributed to the injudicious administration of an inappropriate remedy. Just as is known to have occurred in cases of typhoid fever. When the life of the patient has hung upon such a slender thread that a single small dose of iodide of potassium has sufficed to bring on immediate and fatal collapse. In my own experience this has occurred. The case was that of a widow lady aged about 70, living at East Sheen, whom I saw in consultation with Dr. Hassall and Mr. Cresswell.

Of all medicinal substances, germicides require the most judicious handling. In proof of the truth of this statement I need only refer the doubting reader to the cases which have been published, where rapidly fatal symptoms have followed upon the mere dressing of wounds by their solutions. Many of which are already on record; but I will only cite two, and select them from a foreign source, as recorded in the 'British Medical Journal,' 1881.

It says that two cases of poisoning by carbolic acid are reported in abstract in the 'Nordiskt Medicinskt Arkiv.' One of them, by Dr. J. A. Malmgren,

is that of a child aged 5½ months, who had an eruption, followed by an ulcer, in his groin, which was ordered to be dressed with carbolised oil (8 per cent.). The next day he had vomiting, which was repeated during the night. The urine was 'very dark and foul,' and the child was very sleepy. The carbolised oil was removed on the third day; the child slept almost constantly; the pupils were somewhat contracted. On the fourth day the somnolence ceased, the vomiting was less frequent, and the urine had become much clearer. The child recovered; but the urine retained a dark colour for a fortnight. In the second case, related by Dr. Nordenström, a child one year old had a large fluctuating swelling in the left parotid and submaxiliary regions; it was opened, and pus discharged. The part was dressed with cotton-wool saturated with carbolised oil (1 in 10). About an hour after the application of the dressing, the child had vomiting, which continued through the following day. The urine was of a dark green colour. On the third day, the conditic was about the same, and the breathing was impeded A mixture of equal parts of camphorated oil and olive oil was now substituted for the carbolised oil; but the child died the next morning.

As cases of this sort, though only occasionally reported in the public prints, are most probably not uncommon, and it would be well if we could under

stand their proximate cause, so that we might be able to guard against such untoward accidents, I shall here venture to throw out a few hints. Which may perhaps suggest to the mind of some reflecting reader a theory, which will tend to solve the problem of the exceptional intolerance to particular forms of remedies that certain states of the system occasionally manifest. And, to begin with, I may state that the theory which I have myself formed is that the suddenly fatal results are due to the nervous system of the patients having been, previously to the administration of the fatal drug, so greatly exhausted from the debilitating effects of non-nutrition (from the constituents of the blood, on account of the germs' presence, not being properly prepared for the purposes of assimilation), as to be unable to stand any further depressing influence. This theory I found on the following demonstrable data.

1. Magendie long ago showed that by extracting blood from a healthy animal it was rendered fearfully susceptible to the toxic effects of poisons. He found, for example, that a quarter of the usual fatal dose of almost any mineral poison killed an animal sooner than when the full dose was taken, if not bled before it was administered.

2. That blood, even though abundant, when circulating with its ingredients unfitted for the purposes of tissue assimilation and nutrition, is not one whit

better than no blood at all, is proved by the fact that if a sparrow has its toes slightly pinched after twenty-four hours' starvation, it instantly expires from the depressing effects on its already debilitated nervous system of even that moderate amount of pain.

3. It is my belief that germs, by disorganising the blood—as I showed in my experiments recorded in the 'Lancet' of 1881—reduce it to a nonentity, in as far as nutrition is concerned, and consequently the animal with bad blood in its vessels from the effects of disease-germs is no better off than if it had lost a proportional amount of good blood, and is consequently, like the bled dog and the starved sparrow, rendered incapable of resisting the toxic effects of even small doses of poison. All remedies, be it remembered, are, at the same time, poisons. Their amount alone constituting their right to the respective titles of remedy or poison. This then is, I believe, the philosophic explanation of the intolerance of active remedies occasionally manifested in cases of germ disease.

I now come to the consideration of the destruction of disease-germs within the human body by means of germicides. That this has been successfully accomplished by numbers of us, there can be no doubt, as will be seen in the cases reported further on. Meanwhile I will only here allude to

the cases recorded by Dr. C. G. Rothe ('Deutsche Medicinische Wochenschrift,' 1880, Nos. 11 and 12), and Mr. C. E. Shelly ('British Medical Journal,' April 9, 1881), of enteric fever treated by the administration of carbolic acid and tincture of iodine in frequently repeated doses until apyrexia was produced; and thereafter, at longer intervals, for two or three weeks. The advantages claimed are rapid and permanent subsidence of the high temperature, and of the vascular excitement (the pulse usually falling before the temperature, and often remaining subnormal in frequency for weeks, but not becoming irregular or intermittent); early subsidence of the gastric symptoms (by the beginning of the second week at latest); after which uninterrupted convalescence following.

In a short series of cases of enteric fever which came under Mr. Shelly's observation a few months since, Dr. Rothe's treatment, slightly modified, was put in practice, with results which were not less gratifying. The subjects were young people, their ages ranging from sixteen to twenty-seven years; as none of the cases—with one exception, in which the morning temperature, during the first three days on which it was observed, fluctuated between 104·7° and 105·2° F.—were of more than medium severity at the outset, the patients would probably have recovered under any form of rational treatment, com-

bined with good and careful nursing. But Mr. Shelly was struck by the early and rapid fall of temperature, the retardation and steadying of the pulse, the quickness with which the motions lessened in number and improved in quality, the cleaning of the tongue, the absence of sordes, the early removal of the abdominal pain and tenderness, the refreshing sleep, the comparatively slight emaciation, and the remarkable unanimity with which all the patients agreed in expressing themselves as feeling quite comfortable after the first few doses of the remedy. No increase of temperature was observed to attend the eruptions of the five successive crops of spots which appeared in the most severe case. No complications occurred in any of the cases treated. So that his idea is that the carbolic acid acted as a parasiticide, and, by killing the fever germs, stopped the manifestation of their pathological effects.

Surgeon Worgan, in his report of the health of the 3rd Regiment of Native Indian Infantry for 1879, says that he gave an ounce of water containing 10 minims (? G.H.) of crystalline carbolic acid as often as six times a day with satisfactory results. Salicin he also tried, but he thought that although it reduced the temperature, it had no effect in checking the attacks of the intermittent fever. Probably from the fact soon to be alluded to, namely, that these different antiseptics exert different toxic effects in different

animal fluids, and it is probable that the same principles which control their actions out of the body control them also within the body.

The vitality of disease-germs is very various. Some species seem to be short others long lived. Some resist the action of powerful destroying chemical agents. Others succumb to trifling causes. Syphilitic and malarial disease-germs appear to be the most endurable of all. For once a human constitution has become thoroughly impregnated with either of these species, it may not be able to free itself from them for years—ten, twenty, ay thirty, or even more—after their primary symptoms have manifested themselves. As regards syphilis, this fact is familiar to all ; but as it may not be equally so as regards malarial disease, I may mention that on one occasion I had a patient suffering from hepatic malarial hæmaturia, fifteen years after he left the West Indies (see page 371), where he originally contracted the disease. While I once saw, along with Dr. Phillips, an old Indian officer twenty years after his return from Bengal. Where he had suffered from repeated attacks of jungle fever and dysentery. Who had still an enlarged liver, and was actually seized with a shivering fit while we stood by his bedside. This fit, he insisted, *must* be due to a fresh infection of malaria, as he had not had an attack for several years. A fresh infection, however, was, in

my opinion, out of the question, as he was then living in a healthy part of the Marylebone Road, where ague is unknown—and had been nowhere where it was likely or even possible for him to have contracted the disease, since he returned from Bengal twenty years before.

Now, although this permanency, as it were, of disease-germ existence holds true in the case of syphilis and malarial affections, it is certainly, fortunately for mankind, the exception, and not the rule. For death being the inevitable end of life, all germs die, and with their death their effects usually cease.

In the exceptional cases just alluded to, the permanency of the effects is probably due to the fact that, while in those cases the individual disease-germs perish, the whole species flourishes. Just as colonies of human beings do in suitable localities.

The vast majority of germ diseases, however, may be said naturally to come speedily to an end. Either from the germs' pathogenic action extinguishing the life of their host, or from the food requisite for their sustenance (contained in his body) becoming exhausted, and they themselves being exterminated by starvation, ere they have time to produce their host's vital exhaustion.

In the cases where the patients recover, it may well be asked, what becomes of the myriads of germs which impregnated their tissues and fluids, and pro-

duced the disease ? I suppose that they are eliminated by the various emunctories. For as I showed in the experiments I related in the ' Lancet ' of June and July 1881, the fungi and fungi species of germs, which I injected into dogs' veins, were all got rid of in the short space of forty-four hours. Though in that brief time they had succeeded in destroying the life of the animal. As it is not, however, necessary that the host should die in order that they may be got rid of, and all germ diseases possess more or less of a distinct crisis, I imagine that it is the death and sudden elimination of the dead disease-germs which induce the chain of signs and symptoms usually called the ' crisis.'

This being a new idea of the nature and cause of the so-called crisis of disease, I have deemed it advisable to give my views of the matter in the chapter specially devoted to the ' Fœtor of Disease ' (page 497). Where it is attempted to be shown that the odoriferous perspiration, as well as fœtid urine and stools, which are so characteristic of the crisis of disease, are most probably due to the elimination of dead or living strongly smelling disease-germs.

Were it in our power to imitate all of Nature's methods of destroying germs, we would certainly adopt the system of their artificial calcification ; for that appears to be one of Nature's means of effecting a

spontaneous cure of parasites both large and small. Thus it happens that when a hepatic hydatid, a liver fluke, a filaria, or trichina dies, and it cannot be eliminated, it shrivels up and becomes transformed into a calcareous inert mass. Chiefly consisting of the carbonates and phosphates of lime and magnesia. And just as these large forms of parasites do, so likewise do the minute forms of parasites which we call germs. We know this to be the case at least with both tuberculous and cancerous germs.

FIG. 3.

Encysted cretified Trichina.

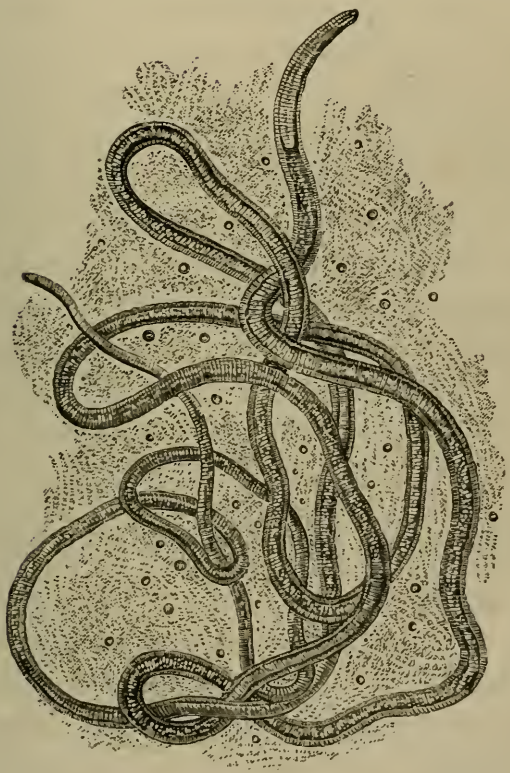
The subjoined woodcut, again, well illustrates the commencing stage of this process of parasite cretification, as it is seen in the so-called tubercular calcification in the lungs of sheep.

This brings me to the consideration of what are germicides ?

The most potent germicides with which I am at present acquainted belong to three distinct therapeutical and chemical groups, and, strange to say, they are all, directly or indirectly, derivatives of the vegetable kingdom. Being of the cinchona, the carbolic acid, and the creosotic varieties. They may be tabulated as quinine, creosote, thymol, eucalyptus oil and gum, carbolic, benzoic, and salicylic acids. I may add to these almost all vegetable gum resins (especially of the empyreumatic pine group), pitch, tur-

pentine, camphor, and such like. All of which have long been looked upon as disinfectants—destroyers of the contagium vivum. But neither salicin nor

FIG. 4.



Strongylus Filaria undergoing Calcareous Degeneration in the Lung of a Sheep.

benzoin are germicides in any sense of the word, for they do not kill germs at all until they are decomposed. Though they are constantly being prescribed for that purpose.

The therapeutical blunders that one occasionally sees committed, by otherwise well-educated men, in consequence of their defective knowledge of physiological chemistry, are not all unimportant. A similarity in name and source fostering the idea of an identical therapeutical action.

Thus it appears from cases reported in the journals within the last couple of years or so that a considerable number of therapeutical errors have been committed from salicylic acid, salicin, and salicylate of soda having been imagined by several to be not only almost identical in therapeutical action, but prescribable in similar doses. While in reality they are not. Indeed are very far from it, so I shall devote a short chapter to their consideration.

Salicylic Acid, Salicin, and Salicylate of Soda.

To begin with, I may at once state that salicylic acid is as different from salicin and salicylate of soda, as sulphuric acid is from sulphur and sulphate of soda, and ought equally carefully to be differentiated in prescribing. For salicylic acid is, like sulphuric acid, a potent poison, and consequently ought only to be administered in very small doses. Whereas the salicylate of soda may not only be given in large doses with impunity, but with advantage. Mr. Watts Parkinson, in the 'British Medical Jour-

nal' of May 7, 1881, while speaking of its beneficial effects in acute rheumatism, says that in the cases in which he administered it, he found that although chloral and bromides moderated the delirium, and procured some little sleep, yet the temperature continued to rise, and the delirium and restlessness went on until salicylate of soda was given. Then there was a daily decline of about a degree in the temperature, and a corresponding improvement in the other symptoms. One gentleman took over a thousand grains of salicylate of soda in six days without any toxic symptoms; and again took, after three days' interval, over seven hundred grains in six days.

In proof of what I say regarding the necessity of not confounding the therapeutical actions of such nearly allied substances as salicylic acid, salicin, and salicylate of soda, I may mention that I have found by a series of carefully conducted experiments upon their respective behaviour towards disease-germs, cultivated in various organic solutions of animal and vegetable matter, that while salicylic acid acts almost invariably as a powerful germ poison, neither salicin nor salicylate of soda, so long as they remain undecomposed, have any such effect. Moreover, chemistry has shown that, instead of being a germicide, salicin is in reality a germ food. Being actually under certain circumstances, like fibrin and sugar, a fermentable substance. Splitting up, under the

influence of sulphuric acid, as well as of the electric current, into, among other things, glucose. Moreover, not only is salicin isomorphic with benzoin, but benzoic acid, as well as carbolic acid, can be prepared from it. The three acids, carbolic, salicylic, and benzoic, are all indeed transmutable into each other, and so easily is this transmutation accomplished, that at the present moment the salicylic acid of commerce, instead of being (as it ought to be) the natural product of the oil of winter green, is in reality artificially manufactured out of carbolic acid. Besides which, I may mention that it can be equally readily prepared in fine needle-shaped crystals from the benzoate of copper (Amer. Chem. Jour., vol. ii. p. 338, 1881).

When salicin is taken into the stomach, it is decomposed in the system, and salicylic acid is one of the products. This can easily be proved by testing the patient's urine with tincture of iron, which gives a fine purple coloration with salicylic acid, but not with salicin.[1]

[1] In testing the urine with perchloride of iron for salicylic acid, it must be done carefully; otherwise the white, cloudy, dirty milky-looking mixture that forms will completely obscure the purple reaction. If the iron solution be poured down the side of the test-tube, and the tube be not shaken, then the reaction will immediately be visible enough; but as it will most probably vanish at once on agitating the test-tube, my advice is to put the test-tube, standing directly on its end, aside for a few minutes, when the white coagulum will subside and the beautifully transparent purple liquid appear on the top of the coagulum. This milky compound, which I have found forms in all normal human urine on the ad-

On the other hand, salicylic acid is a powerful germicide. But only upon those of the animal class. Such as Brownian granules, vibrios, and spirilli. It has no deleterious effect whatever, I find, on bacteria and micrococci. For I have again and again taken two bottles of the same saccharine urine, and while to one I have done nothing, and to the other added a pinch of pure powdered salicylic acid. shaken the bottles well, and then placed them side by side under precisely the same circumstances, in a day or two I have found both equally crowded with torulæ germs. Showing clearly that the deleterious properties of salicylic acid do not act perniciously on the vegetable torula cerevisiæ. On the other hand. again, benzoic acid kills these vegetable germs.

Neither the salicylates of quinine nor soda appear to me to have any deleterious effect on germs whatever, and Müller has made a precisely similar remark regarding the non-antiseptic effects of the benzoate of soda.

Quinine, in the form of the sulphate, readily kills vegetable germs. So does carbolic acid. But carbolic acid, like salicylic, appears to be most deadly to animal germs. I find, however, the effects

dition of perchloride of iron, is not due to the presence of either ordinary albumen nor mucus. Although I have often seen it, I have never taken the trouble to analyse it, on account of time being to me now-a-days a rather valuable commodity. Were I less occupied, I should investigate the point at once.

of these germicides are, to some extent, influenced by the nature of the fluids in which the germs are cultivated. Milk, blood, and urine, all modify more or less their toxic actions on the germs cultivated in them.

The salicylate of soda has recently become a fashionable remedy in hepatic affections as well as in acute rheumatism; but like all other fashionable remedies, I fear it is doomed soon to be discarded. Not however from any want of intrinsic therapeutical merits of its own, but from the injudicious employment of it by its too ardent admirers. Already it has been given so injudiciously, and in such large doses, as to have brought on delirium in some cases, and a comatose drowsiness in others.

It is very easy to detect that an excessive quantity of the drug is being or has been given by the appearance of it in quantity in the urine. After large doses have been taken for three or four days consecutively, salicylic acid may be detected in the urine, sometimes as long as seventy hours after its discontinuance.

The proper dose of the salicylate of soda in hepatic cases is from fifteen to twenty grains three times a day, and the moment head symptoms manifest themselves, it ought to be instantly discontinued.

Benzoic Acid and Benzoates.

There is another remedy to which I desire at this place to call special attention, as it is applicable to the treatment of any form of biliousness and jaundice whenever the staining of the skin produced by the bile-pigment is wished to be got rid of. The remedy to which I allude is benzoic acid and its salts. Benzoic acid was first recommended as a remedy in the treatment of jaundice a quarter of a century ago by a German physician, the name of whom I have forgotten ; but led by his suggestions I have been in the habit of largely employing it, not, however, as he suggested, for the cure of the disease giving origin to the yellow discoloration of the skin, but simply for the removal of the discoloration itself. Having discovered that benzoic acid, especially when in combination with alkalies, in the form, for example, of benzoate of soda, ammonia, or potash, in from ten to twenty grain doses, has a marked effect in causing the reabsorption of biliary pigments from the skin. In fact it acts, as a lady on one occasion graphically described it, by saying that the remedy 'bleached' her, which was a much more philosophic term than what I had applied to it, namely, the title of a whitewash ; for in reality it acts as a bleaching agent by extracting the pigment, not as a whitewash by simply hiding it from view.

Dr. Green (one of my former pupils), who has been resident for several years in Bengal, tells me that, following my suggestion, he often uses the benzoic acid either in six-grain doses in the form of pill three times a day, or in the form of an alkaline mixture (as I usually recommend), and finds it exceedingly useful in clearing away the jaundiced tint of the skin, after the exciting cause of the discoloration has been removed.

To one case in particular he called my attention, namely, that of a man attacked with jaundice following upon delirium tremens, who, he said, after taking the benzoic acid for eight days, was of a perfectly natural colour, and it would have required an experienced eye to detect even the then slight remaining yellow tinge of the conjunctivæ.

The following case may be cited as a tolerably good example of the value of benzoic acid in cases of jaundice from enervation :—

William M——, aged eleven years, labouring under an acute attack of severe jaundice, came under my care at University College Hospital on February 2. The patient appeared to be a moderately developed and very intelligent boy. The jaundiced condition of the skin, his mother said, was first noticed on January 30, only two days before he came to the hospital.

Although the boy had been for some length of

time subject to bilious headaches and vomiting, he had never before (though frequently very sallow) been attacked with marked jaundice. On the present occasion he also complained of headache, but it was unaccompanied either by sickness or vomiting. On examination the liver was found normal in size, and not in the least tender on pressure. The bowels were moderately open, and the stools not pipeclay-coloured. The urine was, however, of a deep orange tint, and the skin of a dark yellow hue. There was an abundance of bile-pigment, but not a trace of bile-acids in the urine.

As the jaundice appeared to be the result of enervation, brought on by over mental exertion, the boy was ordered to be kept from school, and not allowed to read any books (his mother said he was always reading). At the same time three grains of benzoic acid were ordered to be taken thrice a day.

February 9.—The skin was now very much paler, the yellow colour being nearly gone. The conjunctivæ were still yellow, although less so than at last visit. The urine remained unchanged in colour. He was ordered to continue the medicine.

February 16.—The skin was now perfectly normal in colour ; if anything perhaps a shade whiter than natural. Conjunctivæ no longer yellow. Dismissed cured.

In this case no medicine whatever, except the benzoic acid, was given.

As far as my experience goes, benzoic acid appears to be most useful in bleaching the skin in jaundice arising from enervation or from active congestion ; but it is of little or no service until the acute symptoms have disappeared. I am still rather doubtful regarding the mode in which it acts, although it is clear that it hastens the reabsorption from the tissues, and elimination from the body, of the bile-pigment. It thus appears to play the part of a bleaching agent: as my lady patient graphically said, the medicine had bleached her. On one occasion I tried benzoic acid during an attack of jaundice following upon an attack of ague ; but it proved of no service. Indeed, quinine, combined with mercurials, seemed in that case to be the only remedy. When a large dose of benzoic acid is given, hippuric acid appears in the urine. And, as we know, hippuric acid is not a normal constituent of human urine in any quantity whatever, but only normally met with in the urine of herbivora such as oxen, horses, and sheep.

The benzoate of ammonia—an officinal preparation—being very soluble in water (1 in 5) may be employed instead of benzoic acid.

In general I administer the benzoate of ammonia in from fifteen to twenty-five grain doses three times a day in the form of mixture. But sometimes, from

wishing to combine the benzoic acid with a stronger alkali, I order two drachms of benzoic acid to be dissolved by boiling in one ounce of liquor potassæ or liquor sodæ, and then made into a six-ounce mixture, a tablespoonful of which to be taken as a dose three times a day, in a quarter of a tumbler of water. Sometimes again, in order to obtain a clear mixture with less trouble to the dispenser, as benzoic acid is only very slightly soluble in cold water (1 in 300), and exceedingly soluble in alcohol (1 in 4), I add a little aromatic tincture of cardamoms to the mixture, which has the double advantage of making it agreeable to the palate as well as pleasant to the eye.

Mercury and Quinine in Hepatic Diseases.

Both of these substances, separately and combined, have powerful effects in affections of the liver. For while quinine is of itself a powerful vegetable and consequently malarial germicide, mercury, besides its specific cholagogic functions, is a syphilitic disease germicide. Quinine, in combination with mercury, is given with the most marked advantage in cases of malarial hepatitis,[1] and still more so in those rare cases of paroxysmal hepatic hæmaturia referred to at page 374. Crace Calvert has published

[1] It acts, I believe, by killing the germs. Just in the same way as it cures an influenza cold in the head when snuffed up the nose. By killing the disease-germs.

a most interesting series of experiments which he made in order to ascertain the respective powers of different substances to prevent the development of germ life. A few of the more important of his results I herewith subjoin.

Prussic acid, carbolic acid, and cresylic acid prevented fungi developing in a solution of albumen. Quinine, pepper, and caustic lime did the same thing. Chloride of zinc and sulpho-carbolate of zinc, while they exerted no influence on the vegetable fungi species of germs, effectually prevented the development of animal organisms such as vibrios. While, strange to say, the only two substances that he experimented upon, that were found to be inimical to both animal and vegetable germs, were quinine and carbolic acid. The former, the well-known agent in curing malarial disease. The latter, the great antiseptic agent of modern surgery.

In another series of experiments made with gelatin, he found that while with arsenious acid animal germs appeared within two days, no vegetable organisms appeared at all. With protosulphate of iron, again, neither animal nor vegetable micro-organisms appeared. He also found that cresylic acid was the most potent agent in destroying vibrio life. While next to it in order of potency stood carbolic acid, sulphate of quinine, chloride of zinc, sulphuric acid, picric acid, sulpho-carbolate of zinc,

chloride of ammonium, sulphuric acid, and prussic acid.

Common salt, chloride of calcium, chlorate of potash, sulphite and bisulphite of lime, phosphate of lime, hyposulphite of soda, turpentine and pepper had no deleterious action on the animal germs. While, on the other hand, lime, charcoal, permanganate of potash, the phosphate of soda and ammonia actually favoured the production of vibrio germs, and thereby promoted putrefaction (Pharm. Journ., 15th June, 1872).

In addition to these interesting facts, Crace Calvert showed (Pharm. Journ., 15th June, 1872) that decomposing white of egg develops no living germ organisms when exposed under nitrogen, hydrogen, or carbonic acid gases, while it does so freely, under precisely the same conditions, when exposed to oxygen or ordinary atmospheric air. Moreover he adds to this observation the interesting fact that the animal organisms produced under the putrefactive process—which, as I showed, is simply a process of fermentation—when kept in closed tubes exhale sufficient carbonic acid not only to stop their own development, but to asphyxiate themselves, just as a mouse or a man would do if shut up for a sufficient length of time in a closed space with only a limited supply of oxygen.

Pasteur tells us that the germs of chicken cholera

disease are killed by dilute sulphuric acid. While I have noticed the marvellous toxic effects produced upon gonorrhœal pus-germs and spermatozoa by dilute acetic acid. It kills them at once.

Mineral Waters in Hepatic Diseases.

I must not leave the consideration of remedies in the treatment of liver diseases without directing special attention to the valuable curative therapeutical powers possessed by certain natural mineral waters in the treatment of non-organic diseases of the hepatic organ. To wit, our own well-known mineral springs, such as those of the Bridge of Allan, which is a saline aperient, containing 90 grs. of salts to the pint, chiefly magnesian. Cheltenham, which is also a saline aperient, and near it Kingswood, which contains nearly 56 grains of sulphate of soda and magnesia to the pint. Leamington with its 104 grains of chlorides and sulphates, and lastly the Victoria mineral spring (Stratford, Essex) with its 81 grains of salts (chiefly sulphate of soda) to the pint.

On the continent of Europe there are nine mineral springs, much stronger in their hepatic therapeutical action than any of our British ones, which have acquired a very high repute among our continental brethren for the treatment of almost all kinds of liver diseases. In alphabetical order they are the seven

Bads, as they are called in Germany, of Aachen (Aix-la-Chapelle), Carlsbad, Ems, Friedrichshall, Kissingen, Marienbad, and Pullna; and the two *eaux*, as they are named in France, of Vals and Vichy.

I will say nothing more special about any of our British mineral waters, as they are, no doubt, well known to all my readers; but as those of the continent are probably not so well known to them, I shall call attention to their more prominent features, alike as regards position, altitude, and special chemical constituents.

Aachen (Aix-la-Chapelle) is a town of 80,000 inhabitants in Rhenish Prussia. Situated at the height of 450 feet above the sea level, in a volcanic district, at the ends of the Eifel and Ardennes mountains. It possesses a pleasantly cool atmosphere. Which, even in the hot months of July and August, never exceeds a mean of 63° F.

The chief ingredient of its waters is chloride of sodium (common salt). The next in relative proportion is carbonate of soda (washing soda), of which ingredient it contains about a fourth of the chloride, and lastly sulphate of soda (Glauber's salts). Which is, in its turn, in about half the proportion of that of the carbonate. It is thus seen that the mineral base in all these cases is sodium.

Carlsbad, which is situated in the Bohemian

Switzerland, as the district is called in Germany, enjoys the delightfully cool altitude of 1,200 feet above the sea level. So that its atmosphere is, comparatively speaking, cool even in the hottest months of the year, at a time when the lowlands of Germany are parched and scorched by the broiling sun. The chief and most active chemical ingredient in its waters is the sulphate of soda, which it contains in exactly the same proportion as Aix does of common salt. While, again, having only half the quantity of common salt, it has double the quantity of alkaline carbonates, and an equal amount of sulphate of potash, as the waters of Aix-la-Chapelle have of sulphate of soda.

Next in alphabetical order comes Ems. It has a much lower altitude, being only 291 feet above the sea level, and possesses a soft and balmy atmosphere. Ems is a town on the river Lahn. Not very far from its confluence with the Rhine. The chief ingredient of its water is also soda; but this time it is the bicarbonate which is most abundant. Exactly doubling its chloride (common salt). While the bicarbonates of lime and magnesia stand in lieu of the sulphate of soda. Which latter substance only exists in the water in infinitesimal proportion.

Friedrichshall, situated in a valley in Saxe-Meiningen (near Coburg), has a water exceedingly rich

in both the sulphate of soda and the sulphate of magnesia ; contains also a large quantity of sulphate of lime ; besides an enormous amount of common salt. Hence it is an exceedingly active alkaline purgative water.

Kissingen has an altitude of 800 feet, and a mild dry atmosphere. Its waters contain twice as much common salt as Aix-la-Chapelle, while it replaces the carbonate of soda by about an equal proportion of Epsom salts.

Marienbad in Bohemia, again, stands at an altitude of 1,900 feet above the level of the sea, and consequently enjoys the coolest and purest atmosphere of them all. Its waters contain abundance of sulphate of soda, and about equal quantities of baking soda and common salt.

The last of the German mineral springs to which I have to allude is that of Pullna, in Bohemia, which is a strongly bitter saline purgative. Containing three times more of the aperient sulphates than any of the other strongly laxative waters. Although its chief base is sodium, it contains neither the chloride nor the carbonate of that metal. Consequently differs very materially in its therapeutical effects from all the other six waters alluded to. Presently I shall give a comparative table of the chief constituents of these different springs, in order that the reader may see at a glance how their actions on

the liver must vary. But before doing so I shall make a few remarks on the two French mineral waters of Vals and Vichy.

Vals is a town in the province of Ardèche, south of Lyons, highly celebrated for its alkaline springs. The chief chemical constituent being bicarbonate of soda, of which it contains the large quantity of over 30 grains to 16 ounces of water. In addition to this it contains a small quantity of sulphate of soda and chloride of sodium. The next French alkaline *eau* of great repute is Vichy, which is charmingly situated in the centre of France, not very far from Vals, at an altitude of 787 feet above the sea level. Like Vals, it owes the celebrity of its waters to the (still larger) quantity of the bicarbonate of soda (baking soda) they contain.

Although the reader can for himself readily understand, from the constituents of each of these waters, what their therapeutical effects on the human system ought to be, I shall nevertheless point out, somewhat in detail, the comparative therapeutical actions of each of these nine continental natural mineral waters, so that he may the more easily comprehend their mode of action in the general treatment of NON-organic diseases of the liver.

I shall not go into the question of the general action of mineral waters upon the human constitution, but merely direct attention, in a philosophic as well

as a scientfic medical spirit, to the probable mode by which their physiological action on the liver, stomach, and kidneys has a curative effect in hepatic diseases. Alkaline carbonates, chlorides, and sulphates have all respectively specific effects on the biliary, urinary, gastric, and intestinal secretions. An alkaline carbonate, for example, increases the flow of bile, neutralises the gastric juice, and renders the urine alkaline. A chloride acts chiefly in increasing the organic and inorganic substances eliminated by the kidneys, while all sulphates act more or less powerfully on the bowels, and increase not only the number of stools, but the actual total amount of fæces excreted in a given time.

The actions of the nine continental waters on the human system may be gleaned therefore by merely glancing at the comparative table I have drawn out. From it will be learned not only the different ways in which the various waters ought to act, but likewise the varying degrees of intensity with which they will act on liver, stomach, intestines, and kidneys. For, as will be seen by the table at page 231, the waters of Ems are saline as well as alkaline. Those of Vals and Vichy are alkaline without being saline. Those of Pullna and Friedrichshall are not only saline but purgative. While Carlsbad and Marienbad again are not alone purgative and saline, but at the same time alkaline. Those of Kissingen, on the other hand, are

strongly saline, and only slightly purgative; while Aix-la-Chapelle's are not alone slightly alkaline and purgative, but at the same time markedly saline. The manner in which these various chemical properties manifest themselves therapeutically upon the system is interesting.

1. As regards the purgative action of mineral waters. The degree of intensity of course varies with the amount of sulphates they contain.

2. All of them augment, in greater or lesser proportion, the quantity of solids passed from the bowels during twenty-four hours. And the reason of this is very simple. By the purgative effects of the waters the peristaltic action of the intestines is increased, and the food is hurried through and out of the digestive canal before all its nutritive materials have had time to be absorbed from it by the lacteals, and hence more feculent matter is excreted by the bowels than would have been the case had the digestive materials sojourned longer in the intestinal canal, and gone to nourish the body.

3. The saline constituents of the waters by increasing the thirst increase the quantity of liquids ingested, and the more ingested the more solids they dissolve, and as the greater part of the fluid that is drunk is eliminated by the kidneys the urinary secretion both in fluids and solids is proportionally augmented.

4. Although the first effects of alkaline and saline mineral waters are to increase the organic solids passed during the twenty-four hours by the urine, this soon ceases to be the case in those of the waters possessed of purgative properties. In a few days after the continued use of Pullna, Friedrichshall, and Marienbad water, for example, the total amount of organic solids eliminated by the kidneys in twenty-four hours is actually diminished. And this is due to the fact that the nutritive materials are hurried through the intestines, and consequently, from less getting absorbed into the circulation, there is a smaller excess in the blood to be excreted along with the urine; while again, the temporary increase at first is no doubt owing to the elimination of the excess of organic nutritive materials which had accumulated in the circulation before the employment of the mineral waters began.

5. The free use of mineral waters, especially those of the non-purgative class, largely augments the quantity of inorganic solids excreted during the twenty-four hours by the kidneys. The augmentation of sulphates, chlorides, phosphates, &c., being in a direct ratio to the relative proportion of these chemical compounds contained in each ounce of the water imbibed.

6. All alkaline mineral waters render the urine alkaline; with varying degrees of rapidity, in direct proportion to the amount of alkaline carbonates they contain.

From these chemical, physiological, and thera-

peutical data, it will be apparent to every enlightened reader that it is impossible to doubt even for a single moment, far less to deny, the beneficial powers of alkaline, saline, and purgative mineral waters in the treatment of certain non-organic diseases of the liver. I mean by NON-ORGANIC those diseases that are unaccompanied by permanent structural change. Such, for example, as simple hepatic congestions, acute and chronic inflammations. Not such diseases as cancerous tumours, hobnail liver, hydatid cysts, or fibrous deposits. And I think all will agree with me that it is a pity that every spring of the year England should be flooded by hosts of puffing pamphlets extolling to the skies the curative powers of certain continental mineral springs in almost every species of liver disease, when it is evident, from the chemical constitution of the waters so lauded, that such a statement must be untrue. It is indeed to be regretted, if from no other than the mere fact that every ordinarily educated man sees for himself, by the analysis of the waters usually appended to the touting pamphlet, that they contain no extraordinary or uncommon ingredient whatever.

Dr. John Macpherson, in his notes of visits to foreign watering-places, which were published in the 'Lancet' of April 1872, makes some most judicious remarks bearing on this point which I cannot do better than quote here. He says :—

'I had occasion to make the acquaintance of many of the bath doctors, and was much struck with their general intelligence, and pleased with the freeness with which they discussed the operation of their own waters. Few of them, however, appeared to me to take extended views of the subject of balneology; they knew a little of other baths employed for the same complaints as their own ones, and knew something of the baths which they could recommend after their own as after-cures, but their knowledge did not seem to extend much further, nor did they seem to take much interest in the subject in its general relations. One very general subject of complaint among them was, the imperfect selection of cases sent to them for treatment. I thought at first that this might refer only to cases coming from England; but they told me that their own practising physicians and professors constantly sent them the most unsuitable cases.

'Then, when such cases came to them, the difficulty arose, how they were to act: were they at once to declare to the patients that their doctors had made a mistake in sending them there, or might they not discover that, in some respects at least, their own baths might be found useful? Most mineral waters, if they do not happen to be very powerful ones, are applicable to so many different and varied conditions that they easily supply an excuse to a medical man

for making a short trial at least of his own waters on patients who have been sent to him. In such cases they usually endeavour to make up by some more purely medical treatment, or by the use of waters from other sources, for the shortcomings in their own spring in the treatment of a case for which it is not specially adapted. . . . It was freely admitted to me at various baths by the doctors themselves, or proved to me convincingly by the doctors of other baths, that in many cases patients believed themselves to be cured by particular mineral waters, whereas a great portion of the cure depended on the exhibition of medicines. This was more particularly the case with regard to syphilis. Many a patient cured at a sulphur bath in reality owes his cure to mercury, especially in the form of inunction. Again, iodine is used freely in cases of syphilis and of scrofula; and I doubt whether there are any so-called iodine waters the operation of which is not, in certain cases, increased by the artificial addition of that substance.'

This multiplicity of so-called 'Kurs' is a very humbling fact, and one sufficient of itself, it might be supposed, to put an end to all the injudicious laudation contained in the majority of the pamphlets, in some of which we are unblushingly told, in quasi-professional language too, of the transcendental healing virtues of the waters in almost every imaginable complaint—from a simple stomach-ache to an incurable form of

disease. From two mineral-spring pamphlets now on the table before me I extract the following liver affections mentioned in the list of diseases said to be within the curative scope of the waters recommended:—Hepatitis, acute and chronic ; retention of bile ; gall-stones ; enlargements of the liver, and suppurating abscesses. Then follow some more liver diseases with the following high-sounding titles :—Stasis of the circulation ; hyperplasia of the connective tissue ; and hepatalgia. Added to which there is a whole host of stomachal and renal diseases appended. Just as if the waters were an infallible cure for almost every complaint which human flesh is heir to ; and not only are all sufferers invited to come and partake of their healing virtues, but all are equally promised a cure. Even those whose sojourn at the springs may have apparently not at first been attended with the promised results have the cheering assurance given to them that although the benefits derivable from a course of the waters are not immediately apparent they need not despair, as they are nevertheless sure—quite sure—to supervene within six weeks, or at most a couple of months, after the patient's return home. By this wise means none are allowed to leave the springs in a desponding state of mind, but on the contrary all are made to return home—not only if they have as yet received no benefit, but even rather the reverse—in the buoyant hope that 'the good time is coming.' One would almost

fancy that the curative properties of some springs far exceeded, in the eyes of their partisans, those recorded (in the 4th verse of the 5th chapter of St. John) of the pool of Bethesda, where it was only the first who stepped into the water after it was moved by the hand of the angel that received the promise of cure. For, in the monographs alluded to, all are promised a cure, without even having to undergo the fatiguing ordeal of wearisome waiting and anxious watching for the advent of any miraculous moving of the waters. When looked at philosophically, it is easily understood from whence the mineral springs on the continent of Europe have derived their great reputation. It is mainly, I believe, from the fact that the localities in which they are situated supply the great and important want of easily accessible pleasant holiday resorts which the vast majority of our inland-dwelling continental brethren suffer from. From any part of England a two hours' inexpensive railway journey transports an inland-dwelling inhabitant to some one or other of our charming, invigorating, health-restoring seaside watering-places. But, alas ! for the inland-dwelling inhabitant of the Continent there is no such near-at-hand seaside refuge. For him to get to the seacoast requires not only long fatiguing hours of journey, but also a long purse to pay for it. So in the majority of instances he contents himself with the next best thing to a dip in the saline restless

sea. Which is, a dip in the saline placid inland bath. The people on the Continent who cannot afford to go to the seaboard and enjoy the delights and benefits of the 'Wellenschlag,' as they graphically term the 'blows of the waves' of the briny deep, betake themselves with their families during the summer months to some one of the many mineral-water localities; and just as our people talk of it requiring so many dips in the sea to 'set them up,' they talk of their 'Kur' as consisting of so many tumblerfuls, or so many 'Bads.' The continental can no more than the British Pater- and Materfamilias exist without annual refreshing summer holidays, and, while the one set betake themselves to inland mineral springs, the other set, for precisely similar reasons, wend their way to the sea-coast. If Great Britain had not a so easily come-at-able seaboard, we should hear quite as much of Bath, Cheltenham, Harrogate, and the Bridge of Allan as of Carlsbad, Kissingen, Vals, and Vichy. For there, parent and child would be able to enjoy the hygienic curative influences of change of climate, change of scene, change of food, and change of mode of life, which a residence at any and every natural mineral spring furnishes. No matter what the chemical ingredients of its or their waters are. Should any one doubt this, let me remind him of what Christopher Anstey wrote about the watering-place of Bath in the last century. Which is a clever fragment of social

satire on mineral watering-places in general, as it points out in graphic poetry the known attractions which all natural mineral springs possess in a greater or less degree to the desponding invalid:—

> Of all the gay places the world can afford,
> By gentle and simple for pastime adored,
> Fine balls and fine concerts, fine buildings and springs,
> Fine walks and fine views, and a thousand fine things,
> Not to mention the sweet situation and air,
> What place, my dear mother, with Bath can compare?

Dr. Macpherson, in his 'Notes' already referred to, remarks that 'there is now scarcely a considerable village in any tolerable picturesque part of the country, however deficient it may be in mineral waters, that has not its cold and vapour baths, its pine extract, its baths of herbs, and its electrical baths. Most of the new baths now alluded to with *tables d'hôte*, are in fact lodging-houses in which you need not undergo treatment unless you like. A good table is what patients look for in such places, and a good table will confirm the reputation of an establishment more than the most scientific treatment; but it is in this matter of diet that such institutions are apt to be defective. I was repeatedly warned in this sense against becoming an inmate of one of them. I observed generally very few changes from former years as to diet. The table is excellent in most of the larger ones, but there are many crowded baths, as Schwalbach, in which it has not reached the proper standard. English patients

still complain of the want of *tables d'hôte* at Carlsbad, and in Austrian baths, where the physicians endeavour to control the diet of their patients more rigidly than they do in the baths nearer the Rhine.' In my opinion, however, this is one of the greatest of their advantages, for a restricted diet, as shown in the text, is absolutely indispensable in the treatment of many liver diseases. I may add that Kurhäuser, Badanstalten, Trinkhallen, and Trinkquellen, for the cure of every imaginable disease, curable or incurable, by every imaginable appliance, swarm throughout the whole of Germany. Not alone are there natural alkaline, saline, iodine, sulphurous and ferruginous baths and drinking fountains; but there are sand baths at Norderney, mud baths at Driburg, peat-earth baths at Franzenbad, peat-water ones at Untersberg, tar-water ones in the Schwarzwald; grape Kurs at Dürkheim, whey Kurs at Gleisweiler. Besides which there are places for beer Kurs, extract of pine Kurs, juices of herbs Kurs, breathing the air of cowhouse Kurs, condensed air Kurs, pulverised water Kurs, galvanic and electrical bath Kurs, birch-leaf packing Kurs, and so on *ad infinitum*.

In order to show that I am not overstating the case, I subjoin verbatim (with the exception of omitting the prices) a list of the modes of cure held out to the visitors by ONE of the French establish-

ments, in which it will be seen that no less than thirty-eight different forms are enumerated.

Bain simple
„ d'eau de mer „
„ de barège „
„ médicamenteux (suivant les substances)
„ de siège
„ avec douches ascendantes
„ térébenthiné
„ à la sève éthérée de pin maritime
„ *tonique* et *vivifiant*, suivant la formule du docteur P. A. Desjardin
„ *à l'iode naissant*, suivant la formule du docteur J. Bernard
„ à l'extrait d'eucalyptus
„ d'algues avec frictions
„ d'amidon
„ de carbonate
„ de gélatine
Douches simples
„ écossaises, etc.
„ locales simples
Douches locales à vapeur
„ „ „ aromatique
„ „ sulfureuses
Bain de vapeur
„ „ aromatique
„ électrique ou galvanique
Sudation
„ médicamenteuse
Maillots, etc.
Piscine
Maillots à domicile
Inhalations d'air comprimé pour la gorge et la poitrine
Inhalations médicamenteuses suivant les substances
Pulvérisations

Séance d'électricité statique ou dynamique
„ électro-magnétique
Massage oriental
„ simple
Friction simple avec gant
„ au savon

I shall now subjoin my promised comparative analytical table of the active therapeutical ingredients in the waters of the eight most celebrated of the before-mentioned continental mineral springs in the treatment of liver diseases. It has been drawn up from a variety of analyses published by different chemists, and the calculations are made in grains of the substances contained in every sixteen fluid ounces of the waters.

Comparative Chemical Analytical Table of Alkaline, Saline, and Aperient Mineral Waters of the most celebrated Continental Springs.

	Carlsbad	Pullna	Friedrichshall	Aachen (Aix-la-Chapelle)	Marienbad	Ems	Vals	Vichy
Carbonate of Soda . . .	9·0	—	—	—	—	—	—	—
„ Lime . . .	2·0	0·77	10·3	1·4	—	—	—	—
„ Magnesia . .	0·3	6·40	1·1	4·0	—	—	—	—
Bicarbonate of Soda . .	—	—	—	—	13·9	15·0	30·9	37.5
„ Potash . .	—	—	—	—	—	—	2·6	2·8
„ Lime . .	—	—	—	—	6·0	1·7	3·5	3·1
„ Magnesia .	—	—	—	—	5·3	1·5	4·3	3·5
Chloride of Sodium . .	8·7	—	61·1	20·2	15·0	7·7	3·9	4·3
„ Magnesia . .	—	16·66	30·2	—	—	—	—	—
Sulphate of Soda . . .	20·0	123·80	45·5	2·1	38·7	1·4	2.9	3·2
„ Potash. . .	2·0	4·80	1·5	1·1	0·5	0·3	—	—
„ Magnesia . .	—	93·08	39·5	—	—	—	—	—

Be it remembered that this list includes only a mere fractional part of the continental mineral springs possessing almost identical chemical compositions and properties, and the above have been selected merely as types of each class of the alkaline, saline, and aperient waters.

This table of the active ingredients of the eight natural mineral waters of the highest repute in the treatment of liver diseases shows that there is nothing extraordinary, or even peculiar, either about their constituents, or in their proportions, or even as regards their forms of combination. Indeed, on the contrary, these analyses prove that there is neither rule nor order either in the kind of the

ingredients themselves, in their proportion, or in their modes of arrangement, and that, so far from the constituents of natural mineral waters being in the slightest degree peculiar, they are on the contrary precisely such as are to be met with not only in every British druggist's shop, but almost in every English housekeeper's room. To wit, common salt, washing soda, baking soda, together with Epsom and Glauber's salts. Moreover, they show how very different must be the actions of different mineral waters in the same class of disease, and how necessary it is, in prescribing any individual one of them to a suffering patient, to consider the different therapeutical effects of their ingredients as carefully as it is to consider the action of any and every single drug which enters into the composition of a prescription. Nay more, the analyses of these eight celebrated waters still further reveal the important fact that, so far from there being anything specifically curative in any of their constituents beyond the reach of the at home practising physician, he has it readily within his power not only to give every one of their therapeutical ingredients, active or other, in precisely the same form and in the identical proportional combination in which they occur in the natural mineral waters ; but he possesses the yet more important power of being able to alter them at will. To suit the exigencies of any given case, he can not only add to the one and diminish the propor-

tions of the others, but he can altogether omit any one or more of the ingredients just as he chooses. In fact he can not only manufacture a facsimile of the water of any given mineral spring out of the medicinal bottles in his own surgery, by simply mixing their contents in proper proportions with a sufficient quantity of hard spring drinking water, but he can manufacture any new form or combination of the ingredients of natural mineral waters to suit the special conditions not only of the disease, but of the patient's age, sex, habits, and constitution. The only thing which he cannot do, a most important one, is—he cannot combine with his artificial waters the advantages the patient derives from a sojourn at the springs themselves. The immense advantages derivable in the treatment of disease from change of air, change of scene, change of mode of life, coupled with the restorative effects of quietude and freedom from life's cares, are the great factors the at home practising physician has it not in his power to give along with the chemical ingredients of the waters of the natural mineral springs, unless he sends his patient away from home. Again, however, the doctor at the baths themselves labours under equally important disadvantages in treating his patients with natural mineral water. For he is powerless either to modify the relative proportions of its ingredients, or to alter its composition to suit the changing phases of the

disease and bodily condition of his patients. All he can do, and that is but little, is to diminish or increase the daily consumption of the water as a whole, or to refrain from its administration altogether. An overwhelming disadvantage insuperably connected with the treating of any serious form of disease by natural mineral water. In fact, of the two evils connected with the treatment of disease by artificial and natural waters, the former mentioned is, I believe, much the less of the two. For the advantages derivable from the judiciously concocted prescription of the at home practising physician can always be readily supplemented by a change of air, scene, and mode of life, coupled with quietude both bodily and mental, by merely sending the patient a few miles away from his home and its associations. Whereas in no case whatever can the natural mineral water prescribing doctor change the constituents of his prescription to suit the peculiarities of the case.

From these remarks it will be perceived that I look upon mineral waters as powerful chemical compounds, and that in the treatment of serious liver diseases not only must the spring be selected with discrimination, but its waters employed with care. And that whenever it is possible the patient, while taking the water which has been selected as the most suitable in the treatment of his special case, should reside at the spring itself. While on the other hand, when it is

impossible for the patient to avail himself of the advantages of a temporary sojourn at the appropriate watering-place, I think that it is better, in the majority of instances, to concoct for him a prescription containing only such of the constituents of the natural mineral water as are directly suitable for the particular form of liver affection under which he labours than to prescribe for him an inappropriate water. Seeing that we are aware that every particle of mineral matter introduced into the system unsuited to its requirements acts the part of a detrimental foreign substance.

In my opinion, natural mineral waters are simply ready-to-hand made prescriptions, and consequently, like all prescriptions, resemble sharp instruments which may be equally employed in doing good or evil, according to the abilities or the inclinations of their employers. Hence I say that a natural mineral water is no more to be administered thoughtlessly than a dose of black draught or a compound colocynth pill. There is an old poetical adage which tells us that 'fools rush in where angels fear to tread.' Which, in plainer and more homely language, simply means that rashness is the usual accompaniment of inexperience. Just as self-opinion is the equally invariable twin-sister of ignorance. To prescribe for symptoms in cases of severe hepatic disease will, no doubt, after what was said at the

beginning of this chapter, be considered to be reprehensible enough, while haphazard prescribing in doubtful cases will with equal justice be regarded as nothing short of criminal imprudence. For no man has any right to trifle with another man's health, far less with another man's life, by ingenuously adopting the seductive course—on account of its supposed simplicity—of giving a mineral water, under the consoling reflection that 'if it does no good it cannot possibly do any harm.' While in any case it may probably have the advantage of making the patient and his friends believe that the nature and the treatment of the case is thoroughly understood by the dishonest prescriber. In plain language, the man who in such a case prescribes it as a placebo is a quack of the deepest dye. For under the shield of his diploma he commits a practical fraud—and it may too be a dangerous fraud, from its lulling in the mind of both the patient and his friends all suspicion of danger and effectively preventing them from seeking the benefit of further advice, until, when in the end it is sought for, it may come too late not only to retard the course of the disease, but even to save the life of the patient; for, as was said before, hepatic diseases do not, like fevers, as a rule run either a definite or a salutary course. All honourable men will, I am sure, agree with me when I say that it is the bounden duty of every practitioner of medicine—no matter whether he stand at the very

top or the very bottom of the professional tree—when he fails to grasp the true nature of a serious case of disease, at once to ask for a consultation with the most able man he can get. For no matter what his own personal mental calibre may be, or what the extent of his professional experience is, the solution of every doubtful case of disease is invariably simplified by the application of two heads (when they are of the right sort) instead of one.

Baneful Drugs in Hepatic Disease.

While one and all of the foregoing remedies are more or less powerful stimulators of the hepatic biliary secretion, there are a few drugs in our pharmacopœia which have a diametrically opposite effect ; and therefore I must allude to the most prominent of them, in order to prevent their employment in cases of torpid liver, at least when there is no absolute necessity for using them on account of the existence of more urgent symptoms of another kind. The most powerful stopper of the biliary secretion is the acetate of lead ; but opiates, contrary to what is in general believed, act in a precisely similar way on the liver as they do on the kidneys, and greatly retard its biliary secretions. Every one must have occasionally noticed how bilious a patient often looks after he has had for the first time an opiate sleeping draught administered

to him. I well remember after having given a lady lead and opium pills for diarrhœa that she accused me of having made her skin the tint of 'wash leather,' and certainly she did not do so without good cause, as even I was shocked at her sallow appearance, which was so strikingly different from her usually clear white complexion. Either opium or acetate of lead, if given at all to patients labouring under the depressing and enfeebling effects of biliousness, must be given, when possible, in conjunction either with a vegetable or a mineral hepatic stimulant, which in the majority of cases will be found to readily prevent any deleterious action on the biliary function.

Iron is admissible only in exceptional cases; for in almost any form it is a substance which acts banefully in most hepatic cases. In fact a few doses of iron will oftentimes cause the secreting cells of the liver in a biliously predisposed individual to strike work, and a severe attack of biliousness, even amounting in some instances to actual slight jaundice, may be the immediate result. So often indeed have I found iron do harm in hepatic cases that I now never by any chance prescribe it where there is disease of the liver, except in the few rare cases of idiopathic dropsy accompanied with liver derangement, which I shall subsequently take occasion to allude to.

No one has as yet been able to explain the pernicious action of iron in liver cases; all we know about

it is that it has the effect of diminishing the normal transformation of insoluble uric acid into soluble urea by the liver, and that the set of cases in which it apparently acts most detrimentally are those in which the hepatic capillary circulation is most disturbed. To wit, all kinds of congestive and inflammatory affections of the liver. This is not surprising, seeing that iron is seldom or never useful in inflammatory diseases affecting other organs of the body. It is specially objectionable, however, in those of the liver. Even quinine combined with iron ought, according to my experience, seldom or never to be administered in any case of hepatic disease ; but of course there are exceptions here, as there are to every rule. Certainly in none of a congestive, and still less in any of an inflammatory character. For the combination of these remedies, just like iron alone, diminishes the power of the liver to transform uric acid into urea, and hence in many cases they only aggravate the constitutional disturbance.

Dietetics of Hepatic Disease.

I have now to consider the action of food and drink in the treatment of hepatic disease, and I think, if the reader has paid attention to what has been said, in the physiological chapter, regarding the active part the liver takes in the elaboration and transformation of fatty and starchy foods, he will

have little difficulty in perceiving the important part food and drink must play either as banes or antidotes in many of the affections to which the organ is liable. Should the reader not have already given attention to the chapter specially devoted to the consideration of the saccharine, fat-modifying, and calorifying functions of the liver, I think it will be well for him to peruse it (p. 57) before reading what I am now about to say regarding the action of foods and drinks in hepatic cases. Or he will possibly not only fail to appreciate the intrinsic value of the facts brought forward, but even fail to follow the line of argument pursued. Indeed, I believe that the remarks on the action of foods and drinks in liver cases would even be more easily understood if they were delayed until after the subjects of torpid liver and biliousness were considered ; but for the sake of avoiding a great deal of repetition I prefer giving them here. For the general principles I am about to enunciate are not alone applicable to cases of biliousness, but to all cases of deranged liver function, be their cause what it may.

We saw at page 58 that, besides having a biliary function, the liver has also normally a saccharine and a fat-modifying function to perform. So that it may easily be inferred what an important influence an excessive ingestion of either of these kinds of food must have upon the organ when it is in a state of

disease. As elsewhere pointed out, even healthy livers become diseased when an excessive quantity of starchy and fatty foods are given to animals (page 578), and further the fatty and amyloid degenerations thereby engendered are greatly accelerated by heat and want of exercise (page 1014).

It can surprise no one, then, when I say that one of the most fruitful causes of biliousness, in adults as well as in children, is the habitual indulgence in rich fatty, saccharine, and starchy foods. A person *predisposed* to be bilious will sometimes suffer from an attack immediately after one single rich fatty meal. Especially during the hot months of the year, when sufficient bodily exercise is not taken to 'burn off' the excess of hydrocarbons contained in them. For in hot weather, from the body being abundantly supplied with external heat, the animal temperature does not require to be kept up by an excessive internal oxidation of hydrocarbons, as is requisite in the cold months of the year when the animal heat is alone maintained by internal oxidation. Hence in summer, from there being less call for pulmonary and bodily activity, the excess of hydrocarbons admitted into the circulation in the shape of food is, as a natural consequence, not consumed, but simply remains behind in the hepatic cells. Until the secreting cells of the organ become so cram-full of them as to be forced to strike work, and what is

called in milder cases an attack of biliousness is the result.

Not only foods, but drinks, produce the same result. A bottle of heavy stout has often been known, in hot weather, to produce an attack of biliousness in a predisposed individual.

Unfortunately, however, it is not alone mere temporary attacks of biliousness that follow upon an over-indulgence in rich foods and drinks. Their continuous use—especially in hot climates—is a fruitful source of congestion, inflammation, and suppuration of the hepatic tissues.

At one time nearly one half of the liver cases coming home to this country from India were directly traceable to an habitual over-indulgence in rich foods and strong drinks coupled with an indolent mode of existence. Fortunately for the livers of Europeans inhabiting the tropics, the fashion of drinking strong bitter 'Indian ale' at all hours of the day has given place to the less dangerous habit of consuming light French clarets. While at the same time luxurious sofas and wicker lounges have been in a measure abandoned for lawn tennis and foot-ball. And as a natural result not only are much fewer 'Indian liver' cases now met with in London, but the cases that are met with are, in general, of a much milder type than they were between twenty and thirty years ago. Another reason for this

change, however, may be that since the communication with India has become so much easier men run home oftener, and when they get ill, as a rule they return home earlier, and thus greatly diminish the dangers of Indian service. I am sorry, however, to be forced to add—from my own personal observation—that Indian liver cases are still much more common than they ought to be, or would be, I imagine, if more attention were paid to food, drink, and exercise. For careful enquiries among the Indian liver patients who come to me have led me to the conclusion that, notwithstanding the improvement that has taken place in the habits of Europeans resident in the tropics, there is still room for more. As one and all confess that there is still prevalent an habitual over-indulgence in rich, highly seasoned, stimulating food, both by men and women, while resident in climates so hot as to render it impossible for them to take sufficient bodily exercise to use up all the hydrocarbons admitted into the circulation. In fact I believe that it is not so much the heat of the climate as the over-feeding, coupled with the inactive mode of life followed by the majority of English residents in India, which is the *fons et origo* of the greatest number of the hepatic cases there met with.

In this country again, I regret to say that many of the liver cases, especially those met with among women, are due in a great measure to the pernicious

and alas ! nowadays but too common practice of flying to sleeping draughts and soothing mixtures on every trifling occasion. For nothing in the world so effectively and promptly impedes the performance of the hepatic function as narcotics, be they opiates or chlorals. A couple of grains of opium will bring on an attack of biliousness more speedily in most persons than anything else; while its prolonged use will lead to active, as well as passive, congestion of the liver. Even in many cases iron cannot be long given as a tonic without disordering the functions of the liver. As is well known to all men in active practice, many patients cannot live at the seaside for beyond a fortnight or three weeks without suffering either from mere biliousness or from active congestion of the liver.

The stimulating effects of the bracing sea air in general get all the blame of these bilious attacks ; but I have strong suspicions that the biliousness is not *directly* due to the sea air at all, but to an increased appetite, giving rise to an indulgence in a quantity—and quality, it may also be—of food to which the person is unaccustomed when at home. The liver has consequently more work suddenly thrown upon it than it is capable of performing, and, as a natural consequence, breaks down and strikes work. In fact, it acts like a wise horse when overloaded—simply stands still until part of its burden

is removed. This is not only figuratively but literally the case, as I have frequently proved by effectually curing my bilious patients with a day or two's starvation diet.

The two species of food which are in general found to be the most detrimental in the vast majority of hepatic cases are the saccharine-forming and the fatty. Starchy puddings and fat bacon cause more gall-stones in this country, I believe, than all other kinds of food put together, and yet, as far as I am aware, no writer has ever before called attention to this fact. As I go fully into this point in the chapter on the etiology of biliary concretions, I shall say nothing more about it now, but refer the reader to page 580, where he will find that the effects of climate are also considered in connection with the formation of the pathological fatty product cholesterin.

In the treatment of all hepatic cases I attach much importance to diet—and no wonder, seeing that the liver has a sugar-manufacturing, a fat-modifying, and a bile-forming process daily and hourly to perform, and when either one or other of these functions is out of order, the kind of food upon which the function specially acts cannot be given to the patient in excess, or even in ordinary quantity, with impunity. For, as is well known, every organ acts better upon little than upon much, at all times and under all circumstances.

Moreover, it is a peculiar fact that bilious patients are more frequently found to be the subjects of dietetic idiosyncrasies than others. A food that will be palatable to one is in many instances found to be obnoxious to another, without the slightest apparent reason. In illustration of this I will cite a few most telling examples.

Thus, many bilious persons cannot touch either milk or eggs without being made ill. Others can indulge freely in milk, and not in eggs. While again to some the white of the egg is an agreeable food, and yet the yolk is but little better than an emetic.

Even as regards tea and coffee, bilious people show equally strange idiosyncrasies. Some can drink both with pleasure and impunity. Others again find tea alone palatable, coffee acting on them not only as an emetic, but as a purgative. To me the most extraordinary thing of all is that I have a lady patient—for whom I have occasionally prescribed during the last twenty years—and she has told me not merely once, but many times, that a single cup of coffee, with or without milk, induces in her a diarrhœa within an hour after partaking of it, while chocolate has a diametrically opposite effect. So much so indeed that a cup of chocolate instantly stops the diarrhœa occasioned by the coffee. This has always been, and still is, to me a dietetic puzzle. This same lady, who is now forty-five years of age,

married, and the mother of five healthy children, is an inveterate tea-drinker. I say inveterate, for she will drink tea when she has the chance at any hour of the day or night. She begins with an early cup of tea before she rises. She drinks tea at breakfast. She has tea in the afternoon. She takes it again after dinner, and it is no uncommon thing for her to drink another cup about twelve o'clock at night before retiring to rest, with apparently nothing but a beneficial effect. How then, I ask, is it that a small quantity of caffein upsets her system, when a large quantity of thein—a substance almost identical in chemical composition and physiological properties —does her good? I know not, except it be owing to the difference in the aromatic ingredients in the two beverages. Just in the same way that brandy, whisky, gin, and rum produce—as was shown by the late Dr. Edward Smith—different physiological and pathological effects; notwithstanding that the supposed most active base of them all is precisely the same—namely, alcohol. The difference in their mode of action, as Dr. Smith suggested, is probably due to the aromatic principles they respectively contain; and the same idea is the one I entertain with regard to the difference in the action of chocolate, tea, and coffee on the human system.

With shell-fish foods equally anomalous effects are met with in bilious people. One can, in modera-

tion, partake of all kinds. Another can only eat one or two kinds. Some will sicken at the mere flavour of a lobster, or at the sight of a raw oyster; while the same individual will consume (what we call) an indigestible crab with perfect comfort and relish. In fact the same peculiarity of constitution which produces a tendency to disordered hepatic functions gives rise, I believe, to the most anomalous dietetic idiosyncrasies. So that one has to be more than usually particular in prescribing any one particular form of diet to a bilious patient. But, as a guide to the reader, I may say that, as a general rule, the following principles may always be acted upon, at least until the special peculiarities of the invalid have been ascertained.

Firstly. All fatty matters are to be reduced to a minimum, and butter and bacon totally avoided in the vast majority of hepatic diseases.

Secondly. All salted and spiced foods are to be shunned. Such as corned beef, ham, sausages, bloaters, Finnan haddocks, and all other kinds of salted fish.

Thirdly. All river fish are to be eschewed. Salmon, trout, and eels more especially. On the other hand, white sea fish in the shape of cod, sole, turbot, whiting, smelts, and dabs, may be taken with impunity; but mackerel, pilchards, fresh herrings, and sprats, ought not, as a general rule, to be partaken of.

Fourthly. Pastry—whether as pie or tart crust—

dumplings, plum-puddings, and all heavy starchy and sweet dishes, are to be avoided. Whereas plain arrow-root, sago, tapioca, and such like light and pure farinaceous dishes, may be moderately indulged in.

Fifthly. The lean of mutton and beef is a better animal food, because more nourishing and digestible, than poultry or game. Pork, lamb, and veal, being less nutritious, are to be avoided.

Sixthly. Beers and porters, port, madeira, and sherry, are more likely to do harm than weak gin, whisky, or brandy, and claret, hock, or champagne.

All stimulants should be given in small quantities, diluted with sparkling alkaline natural or artificial mineral waters. Iced in summer, but not in winter.

As champagne plays a not unimportant part in the treatment of all the more exhausting forms of liver disease, I shall take the present opportunity of ventilating my views on this universally appreciated vinous beverage. For I wish to change, if possible, the present pernicious English habit of drinking sour wine disguised under the name of *champagne sec*, under the mistaken notion that it is good and wholesome wine which has become naturally dry with age. While in reality it is no such thing, but *tastes dry*, simply because it is *sour*. I say sour, for the various degrees of dry, very dry, and extra dry (*sec*, *très sec*, *et brut*) champagne are simply wines of different de-

grees of acidity—sourness. If any reader doubts this, let him for himself make the experiment of dipping a piece of blue litmus test-paper into his fine (?) dry wine, and (if not already aware of the fact, which in all probability he is not, or he would not have a single drop of the liquid within his doors) I promise that he will open his eyes wide with astonishment at the tint the paper will assume. Vinegar, pure strong wine-vinegar, will not bestow upon it a brighter red tint—and why ? Simply because the flavour which he ignorantly imagines is due to 'dryness' is, on the contrary, due to the presence of acid. And the secret is simply this. 'Dryness,' as it is absurdly called, is the product of age. It is in fact due to the slow transformation of the sugar in the bottled wine into alcohol—as takes place in the twenty, thirty, and forty year old port. But it does not pay the wine merchant to keep his champagne until its saccharine matter has been transformed by fermentation in the bottle into alcohol and the sweetness of the wine has consequently disappeared. So he adopts the speedier course of getting rid of the sweet flavour of the wine by setting up the quick acetous instead of the slow alcoholic fermentation. Which has the effect of destroying all the saccharine matter contained in the bottled wine in a few months. Or he adopts another equally effectual course, of adding less than four instead of, as he ought, eight per cent. of syrup to the

wine at the time of *dégorgement*. In fact there are many ways, well known to the trade, of 'spoiling' champagne to suit the ignorant depraved taste of the English consumer. I say English, for no other nation has as yet been found foolish enough to swallow sour wine under the delusion that it is drinking good sound dry wine from finding the words *sec*, *très sec*, or *brut* on the labels of the bottles.

Of course some of my readers will think this very strong language ; but let me tell them that not a syllable of it is too strong, and if any one doubts the truth of what I say, and thinks he knows a deal more about the matter than I do, let him take the trouble to make enquiries of a champagne wine merchant—not an ordinary English wine merchant, for most probably he will be as ignorant of the whole matter as the reader is himself, but a French wholesale champagne wine dealer—and he will soon discover that every word I have said is not only perfectly true, but not even exaggerated. The following anecdote will show the ideas of a Continental champagne dealer regarding the Englishman's knowledge of good champagne.

All my champagne I import myself, and, as my personal friends know, it is A1. The gentleman, whom I in general deal with, comes to England occasionally, and on one occasion when he called upon me and got an order, I observed that, in booking it, he wrote after the name of the wine the word CONTINENTAL,

and not only so, but carefully underlined it. On seeing him do this with an air of the most perfect *sang-froid*, I exclaimed in a voice of surprise, 'Why have you written the word "Continental" after the champagne? Is all your champagne not *Continental*?' To this he immediately replied, and that too with an air of sweet innocence—'Oh! no. We never now send any Continental wine to England.' 'What on earth do you send then?' exclaimed I in breathless astonishment. He smiled, and answered—'Spoilt English champagne.' Being more bewildered still, I slowly repeated his words, 'Spoilt—English—champagne! What do you mean?' 'Oh! Doctor Harley, don't you know that all the dry champagne is specially prepared for the English market? We can't sell a single drop of it on the Continent; for nobody will drink such stuff. It's quite sour.' Seeing my consternation, I presume, he quickly added—'We never send it to you. Your champagne is what we drink ourselves. It's true champagne—none of the *très sec* stuff.' 'But,' said I, 'all good champagne is slightly acid on account of the carbonic acid it contains.' 'Yes. That is perfectly true; but it's not sour, which so-called dry champagne is. You're a chemist. The first time you have the chance, dip a piece of blue litmus paper into the two wines, and you will soon see the difference. While the Continental one will yield a faint pink, the Eng-

lish one—that is, the sour, which you call dry wine—will immediately turn the paper crimson, just as sulphuric acid would.' Here was a piece of important information for me, and now I shall proceed to give a little further information which may perhaps prove almost equally interesting to the reader.

The very next day I was called by Dr. Macaldin to see an old rich bachelor, suffering from a violent bilious attack, accompanied not only by vomiting, but by diarrhœa. On finding that it was brought on by his having made a hearty dinner of *a pound of salmon steak* and a bottle of champagne, without partaking of anything else, and my wine merchant's information regarding 'spoilt English champagne' running in my head, I mildly asked if the champagne partaken of was *sec* champagne. The prompt reply was 'Oh! yes—the very best *très sec* that can be bought. It could do me no harm.' 'I am not so sure of that,' said I. 'Perhaps the wine is sour.' 'Sour!' said he. 'I never drink common 40*s*. trash. My champagne stands me 84*s*. per dozen, and is as dry as an old bone, and without a particle of acidity in it.' 'Are you quite sure of that?' said I. 'Yes, perfectly sure. You can take a bottle of it home with you and test it.' 'If you will allow me,' said I, 'I'll test it here. I have a piece of test-paper in my pocket, and it will soon tell us whether the wine is sour or not.' 'Then do so,' said he; 'but wait a

minute. You shall have a clean glass and a fresh bottle.' While this arrangement was being put in execution, I took out the litmus paper from my purse, walked across to a side table on which I saw the cruet-stand, took out the vinegar bottle, and nearly half filled a wine-glass with its contents. Then poising a piece of blue litmus paper between my finger and thumb, I said, 'Do you see this? Now watch the result.' The end of the paper was plunged into the vinegar, held there for a second or two, then withdrawn. 'There! you see it has turned from blue to red; that shows that vinegar is sour.' 'Of course,' replied our confident patient; 'but that's sour vinegar, not fine old dry champagne.' The butler had brought in a fresh bottle, and a couple of clean wine glasses for Dr. Macaldin and me. Pop went the cork. Gurgle-gurgle went the sparkling effervescing fragrant wine into the glasses. I took one of them up in my hand, held it straight out in front of me, close to his bedside, and quietly waited until all the froth had disappeared from its surface in order to make sure that the chief part of the carbonic acid had escaped. Then poising as before a fresh strip of blue litmus paper between my finger and thumb, I looked significantly at the sanguine smiling face of the patient, and with marked deliberation quietly and slowly dipped the strip of paper deeply into the wine, let it remain there for a few seconds, and

then as deliberately and slowly withdrew it. What was the result? Nothing more or less than that when it was placed side by side with its fellow, that had been dipped into the sour vinegar, both were found to be nearly equally crimson in colour. The patient's face no longer wore a smile. It was now as proportionately long as it had before been broad. His eyes stared motionlessly, he gazed into vacant space. Not a syllable escaped from the lips of either of us. Then, suddenly, as if a bright thought had struck him, he shouted to the servant to bring him his writing-case. It was soon placed beside him on the bed, and immediately opened. When, after rummaging about in it for a little time, forth came a piece of folded paper, and, tossing it to me, he, with a gruff voice, exclaimed—'Read that.' I took up the paper. It was a receipted bill. I read it over, and from it learned the to me interesting fact that our patient's *très sec* champagne was put down at ninety shillings a dozen, and that six shillings a dozen had been deducted for cash payment. So that the sour trash which he had ignorantly consumed as 'dry wine' had, as he said, cost him 84*s*. per dozen. This proved a memorable night to him; for, as Dr. Macaldin afterwards told me, the little chemical experiment I performed upon the beautiful dry(!) wine had kept the poor patient awake the whole night. He could not get a single wink of sleep; for the colours blue

and red, and the words *sec* and sour, as he said, haunted him like a nightmare. I have only to add, that from that day to this I believe this gentleman has followed my example, as many others of my friends have done, and left the drinking of *très sec* to those happy mortals who enjoy a life in a fool's paradise, where ignorance is bliss, and turn their poor stomachs into pickle-jars while drinking different grades of wine vinegar under the sweetly seductive titles of *sec*, *très sec*, and *brut* champagnes—specially manufactured for their depraved tastes.

Having drunk, I believe, either on the Continent or in England, nearly every natural (!) species of sparkling wine manufactured in Europe, I think I am justified in thus venturing to express an opinion on the merits or demerits of our modern forms of English champagne, and as an additional, perhaps not uninteresting, piece of information, I quote the following notes, which I wrote on the fly-leaf of my passport book, on August 17, 1855, while exploring the underground champagne cave cellars of Jacquesson et Fils, at Châlons-sur-Marne, along with my old student friend Mr. J. T. Poyser—now one of the partners in the well-known firm of Allsopp and Sons, brewers, at Burton-on-Trent. I quote the notes exactly as they are written; for even as they are, with their imperfections, they are more valuable on account of their exactitude than if I were to rewrite them in improved language.

'August 17, 1855. Two millions five hundred bottles in cave. In 1848 there were four millions.

'The wine when pressed from the grapes is divided into three kinds. From the first pressing comes the Crème de Bouzy. From the second the Fleur de Sillery. And from the third the Sillery Supérieur. The remainder goes for the common sort of champagne, sold at two francs and a half per bottle.

'The expressed juice of the grapes is allowed to ferment during fifteen days. It is then run off into casks, in which it is kept from two to six months, and then bottled. The bottled wine is now kept from two to nine years, according to quality, before being "dégorged." After being dégorged—that is, freed from all the deposit accumulated in the neck of the bottles during the fermenting process (which goes on more or less the whole time) while the bottles are all kept standing in racks with their mouths downwards—a varying quantity of a syrup, composed of sugar-candy dissolved in wine is added to each bottle. The quantity of syrup added depending upon the country for which the wine is intended.' (Thus at the time when these notes were taken) 'the wine intended to be consumed in France and England is treated identically with 8 p. c., and it is considered to be dry (*sec*) wine. That for Prussia has 13 p. c. added. While that for Russia (which is considered a sweet wine) has 16 p. c. of syrup put into it. That is

to say, twice as much as is added for France and England.' A supplementary note says :—'When the wines are not naturally strong, some alcohol is added to them before the first bottling.'

Although these notes were hurriedly taken by me as those of a mere tourist, on the fly-leaf of my passport book, and without the remotest idea that I should ever publish them, I have no doubt that, even imperfect though they be, they will stand the touchstone of mental analysis in point of exactitude. One word more on the subject of champagne, and I have done with it. And that is to explain to my reader the meaning of the word *brut*, which one sometimes, but luckily not often, sees upon the labels of the champagne bottles handed round at dinner parties by men who buy their wines according to 'brand' and not according to quality, as judged of by nose and palate. To this French word *brut* I should add an *e*, and transform it into an English word which exactly typifies its true character—'Brute Champagne.'

In the eye of the champagne trader the word *brut* simply means a wine that has been left to itself to undergo fermentation ; but this, which might appropriately be called natural champagne, is poor and sour. So that even the so-called natural *brut* champagne has to undergo a process of 'doctoring' at the period of *dégorgement* to suit it for the English palate.

Hence even it also is artificial. In fact all refinements of human taste are simply artificial acquirements which have become popular, and at length got gradually stereotyped by fashion. In proof of this, I shall now show how the most disgusting drinks and foods are made by fashion not only palatable, but exquisitely delicious ! The Fiji islanders, for example, drink with *goût* and intense delight what appears to us in the light of a disgusting concoction, called 'Cara' or 'Kava,' which Admiral Sir Henry Denham tells me, from his own personal observation, is prepared as follows :— The women sit down in a circle, and chew the root of the *Piper methysticum*. Spitting the juice, mixed of course with their own buccal secretions, into a bowl placed in the centre of the group. Water is then added in proper proportion, and the mixture allowed to stand and ferment for two hours, at the end of which time it is ready for drinking.

To us this appears a loathsome beverage ; but to the Fijians it is a 'dainty dish fit to set before a king,' and is in reality set before their king. For he has a great dish full of it brewed all for himself. (A huge specimen of these Kava bowls may be seen in the Museum of the Kew Botanical Gardens. It is much deeper, and nearly as long as an adult's coffin.)

So much for funny artificial drinks. Now for even a more strange kind of artificially manufactured food. In Kane's 'Tour in North America,' I came upon

the following receipt of the Chinook tribe of Indians for the preparation of a savoury dish of olives.

A hole is dug in the ground near the entrance of the family mansion—the hut. About a bushel of the finest acorns are then put into it, covered over with a layer of grass, and then the hole filled in with about half a foot thick of earth. Now the work of cooking begins. From this time henceforth, for the next four or five months at least, every man, woman, and child in the family urinate into the hole, and in due time the acorns become saturated and softened, pungent, and odoriferous, and are then partaken of as one of the finest of all earthly delicacies. Such at least is the opinion of the refined-tasted Chinook Indians. Can we then feel the least surprised that the educated, highly civilized, though, in wine knowledge, ignorant Englishman should equally prize and relish sour champagne? He is only to be pitied, not to be blamed any more than the Fiji islander or Chinook Indian. On the wise philosophic principle, be it said, that what is one man's meat is another man's poison. we find the British matron priding herself on the nice flavour of her high (*putrid!*) game, the Spanish lady on that of her *rancid* salad oil, and the German Hausfrau glorying in the *geschmack* of her stinking cheese (=Handkäse).

In conclusion, then, my advice is, when ordering champagne for an invalid, to tell him to get wine that

is neither labelled *sec*, *très sec*, nor *brut*, but wine such as is drunk in France, which is not 'sweet champagne' —such as is sent to Russia—but really good wholesome palatable wine.

For those of my readers who are not physiologists, I may add that Russia is supplied with sweet champagne solely on account of its cold climate. The sugar in the wine supplying to the body hydrocarbons which give to it warmth by being oxidised in the system. On speaking to my friend Mr. Poyser on the subject recently, and reminding him of the information we received in the champagne cave at Châlons, he told me that all the ale his firm (Allsopp and Sons) brewed for the Arctic Expeditions in 1852–3 and 1875–6 was made not only strong in alcohol, but rich in saccharine matter—containing 8·62 per cent. of alcohol and 14·3 per cent. of sugar. That is to say about twice as strong as the best Burton ale. The extra amount of saccharine matter being added solely with the view of supplying the Arctic explorers with materials to keep up their animal heat.

In 1881 he opened a bottle of it in order that I might satisfy myself of its qualities by personal observation—and I found it not only in splendid condition, but just such as I should consider admirably adapted for furnishing to the blood in a most palatable form hydrocarbons for oxidation, and the consequent development of animal heat. We, living in this

mild climate, do not require saccharine alcoholic drinks, as the inhabitants of cold countries do. But neither do we require sour ones like *sec*, *très sec*, and *brut* champagne. What we want is neither sour nor sweet wine, but a good sound wholesome beverage precisely the same as the inhabitants of France drink themselves. In fact 'Continental champagne.'

It must be borne in mind that not every change which is produced in the saccharine matter of wine by the action of ferments is true fermentation, for some are merely forms of oxidation. For example, as was shown by Boutroux (Compt. Rend. 91, p. 236), when the ferment Mycoderma aceti is added to a solution of glucose, gluconic acid is formed by each molecule of glucose combining with a single atom of oxygen; and this is not fermentation at all, for an exactly similar change (as was shown by Maumené, Compt. Rend. 91, p. 331) occurs in the oxidation of glucose by cupric acetate and mercuric oxide, where no ferment exists. Cane sugar again in the same sort of way without fermentation (as was pointed out by Reichardt, Centralblatt, 1880, p. 559), is converted by bromine into gluconic acid, glucose, and gum. All of which are acid transformations of saccharine matter quite independent of fermentation. So there is no difficulty in understanding how it is that manufacturers find it so easy by a variety of different processes to annihilate the sweetness of the sugar in

their champagne, without patiently waiting for its slow normal transformation into alcohol.

This reprehensible process of preparing champagne for the English palate is an exactly analogous system to that adopted by our farmers of feeding up lambs to the size of sheep in the course of the first year of their age, and selling their flesh under the name of mutton, of which it possesses neither the flavour nor nutritive properties. For mutton does not come into existence until the animal is at least three years of age, and does not arrive at perfection until it is six. However, as the object of the farmer is simply to get money, so long as he can get ignorant people to imagine that they are eating mutton when they are only eating big lamb, he supplies them with it.

General Remarks on Special Forms of Treatment.

Having so far explained the therapeutics and dietetics of hepatic diseases, I shall now add a few words on some special forms of treatment. Which I feel sure, from personal experience, will not fail to be appreciated by the reader who feels at all interested in the subject. So to commence with I may just as well tabulate a few rules for general guidance. Which may of course be modified according to the special circumstances of each individual case.

1. In the early stages of all cases of acute and severe hepatic diseases, an avoidance of rich nitrogenous foods is indispensable. No solids whatever should be given, and nitrogenous food only in the shape of beef-tea and milk. Slops is the word which most graphically defines the suitable diet, and of those, as everyone knows, there is an endless variety to select from. So I need not waste time by enumerating them. Fruits when in season, such as grapes, oranges, and strawberries, will, if the patient can take them, do good.

2. Let the patient drink freely. The more he drinks the better. For it favours elimination by kidneys, skin, and lungs. In all cases attended with pyrexia, give iced beef-tea and milk. The juice of a fresh lemon, or better still of a lime, in a bottle of aerated water, dilute raspberry vinegar, or anything else of that kind. Oxygenated water I have found both palatable and refreshing to the patient.

3. If the temperature of the body be high, reduce it by the application of ice-bags to the extremities, or even to the abdomen ; but never to the chest, for fear of producing complications. Always take care that the ice-bags are changed from place to place, or the skin may be frost-bitten. An accident which occasionally occurs when the freezing mixture is kept too long on one place.

The plan of placing the patient in a cold water bath is not nearly so good as the applying ice-bags. First on account of its exhausting effects, and secondly the trouble. When patients can afford it, a water mattress, in which the water can be frequently changed, may be used.

In the treatment of some of the more common forms of pyrexial disease, this plan has been successfully adopted at University College Hospital, the Manchester Infirmary, and elsewhere. Where the above methods are inapplicable, begin by putting the patient into cotton sheets. Dispense with everything woollen. Sponge the whole of the body piecemeal (keeping all covered but the part being operated upon) with vinegar, milk, or eau de Cologne and water, twice or thrice a day. For it is not only a cooling but a refreshing process to a feverish body.

4. When the pulse is high, and the patient's strength will bear it, reduce it by half or drop doses of the Pharmacopœial tinct. aconiti, or twenty minim dose of tinct. digitalis, given every hour or two ; but stop the administration of either drug the moment the pulse falls to 80. To be resumed if and when necessary.

5. Vomiting subdue by cold effervescing salines. The citrates of soda, potash, magnesia, and of caffein, will be found useful.

6. Diarrhœa control by chalk mixture, krameria, kino, and other such like forms of astringents.

7. Hæmorrhage. As the causes of intestinal hæmorrhage, which occasionally takes place in the course of cases of liver disease, are very various—occurring as it does in perforating gall-stones, contagious jaundice, and in chronic as well as in acute atrophy of the liver, a correct diagnosis of its cause must necessarily precede the selection of the plan of treatment. But in general terms it may be said that while, in cases of traumatic hepatic hæmorrhage, ice-bags may be applied to the abdomen, in most of the other forms the repeated administration of

℞ Pulv. Aluminis . . .	gr. x.
Acidi Sulph. Aromat. . .	♏ xxv.
Ferri Sulphatis	gr. j.
Syrupi Tolutani . . .	ʒ ij. M.

will in general be found of great service. A new form of hæmostatic introduced from America is Hazeline (the active principle of the bark of the witch-hazel), which in doses of from thirty drops and upwards is likewise efficacious.

8. When cerebral symptoms appear, apply, without a moment's delay, a freezing mixture of salt and pounded ice, in a waterproof bag, to the head. And never forget to keep the part of the bag in contact with the skin moist with water, as dry waterproof

cloth is a bad conductor of cold. Liquor ammoniæ acetatis is useful in large doses. So also is James's Powder.

9. In order to procure sleep, NEVER give an opiate, if it can be avoided, in consequence of its tending to stop the action of the liver, as well as that of the kidneys and bowels. First try the bromide or chloride of ammonium, and, if they will not do, chloral, lupuline, or hyoscyamus.

10. Keep the room as quiet as possible, the patient from speaking, and garrulous friends from approaching him. Stop his ears with cotton wool, and shade his eyes from light by dark blinds or a properly arranged screen. whenever there are the slightest cerebral symptoms present.

11. The temperature of the room should, when possible, always be maintained at between 60° and 65°. In summer, the reduction from a higher temperature to the above is most easily effected by calico screens, moistened with carbolised water, being placed opposite and near to the open windows and doors. While, in winter, it is best raised to it by a briskly burning open fire. The patient's eyes being protected from its glare and flicker by a suitably arranged carbolised water screen, which has the additional and immense advantage of keeping the air in the room deodorised, to the benefit alike of patient and nurse.

12. Perspiration encourage by sweet spirits of nitre, spirit of juniper, squills, and digitalis ; for the skin is a potent eliminator of biliary substances.

13. The kidneys keep equally freely active. Fortunately in this case the same medicines which increase the perspiration also augment the urinary excretion.

14. The bowels ought in all cases to be kept open by the administration of simple purgatives. But at first it is well not only to clear out the intestines of all fæcal matter, but also to aid the action of the liver and kidneys by freeing them as far as is possible from congestion, by employing a remedy which possesses in itself this triple power of action—calomel, or, if not calomel, at least mercury in some form or other. The more plethoric the patient, the bigger the dose. To the weak and emaciated should be given as small a one as will suffice to produce the desired effects, and nothing more.

15. As all the excretions are peculiarly offensive in cases of hepatic disease, they ought never to be allowed to remain in the room. As even the perspiration is in many cases not only unpleasant to the bystanders, but to the patients themselves, if they perspire freely, have the body sponged and the linen changed twice a day. In all the cases subsequently to be described in which jaundice occurs associated with fevers, pyæmia, and septicæmia, the

perspiration is so offensive that the sponging should be done with tepid carbolised water, Condy's fluid, or some other form of disinfectant. The cleansing of the cuticle has the great advantage not only of mitigating the disagreeable odour, and soothing the feelings of the patient, but, by encouraging the perspiration and elimination of bile matters, of favouring convalescence.

16. For similar reasons, it is not only well to have deodorisers placed about the room, but even under the patient's bed.

17. When there is acute inflammation of the liver, apply either leeches or the cupping glasses. But in no case of hepatic germ-disease extract blood. In some cases it is even more likely that transfusion than depletion may be necessary in order to save the life of the patient. A new and simple method has recently been proposed, and is said to have been successfully practised in Germany. It is simply to inject blood into the patient's abdominal cavity, and leave it there to take its chances of absorption by the peritoneum of the abdominal walls, intestines, and other included organs with which it may come into contact.

18. A dry and brown tongue is an indication for alcoholic stimulants, even when the patient is delirious. For they sometimes both clean the tongue and mitigate the cerebral symptoms. Carbonate of

ammonia in ten grain doses is an excellent stimulant in cases of liver disease with delirium, especially when given along with the liquor ammoniæ acetatis. In all cases where there is active delirium, keep the patient's head high. Where there is a tendency to syncope or somnolence, low.

19. Coffee and tea are excellent stimulants in hepatic diseases, and may be administered cold during the very height of the fever at the time all alcoholic stimulants are contra-indicated. The proper time to use alcoholic stimulants is during the convalescent stage, when wine—even strong wine like port, and burgundy, or sound—not sour—champagne, is in all cases better than brandy. For the wine nourishes as well as stimulates.

20. In bad cases, the food and the medicine should be given in divided quantities alternately every half-hour. But if the patient chance to be asleep—unless he happen to be in a continual state of drowsiness—he is not to be waked to get either his medicine or his food, as refreshing sleep is better than food, drink, or medicine—at any rate until he awakes.

21. Hiccup, though scarcely ever alluded to in books on liver diseases, is occasionally a most distressing symptom to the patient. It occurs only in very bad cases, generally in the aged, and in almost all of them it is found that the hepatic disturbance is

associated with some form or other of germ-disease, pyæmic, septicæmic, or typhoid.

The treatment I have found most successful is to administer hydrocyanic acid or a few drops of chloroform along with belladonna, aconite, or morphia. But as the hiccup sometimes resists these, I propose, on the next occasion, to try the effects of the inhalation of the following mixture, which I have found exceedingly beneficial in the subjugation of violent attacks of spasmodic asthma.

℞ Amyl Nitrit. . . . . ℥ss.
Sp. Æther. Sulph. . . . ℥vj.
Sp. Ammon. Aromat. ad. . ℥j. M.

A few drops are to be put on a handkerchief at a time, and the vapour gradually inhaled.

No sooner is the crisis passed, especially if the liver affection should have been in any way connected with epizootic or miasmatic disease-germs, than it is necessary to administer generous diet, and likewise tonics.

Having finished with preliminaries, after a due consideration of which the reader will have no difficulty in following the lines of argument, or of understanding the true nature of the facts upon which the subsequently to be described plans of diagnosis and treatment of liver affections are based, I now turn to the consideration of the clinical part of my subject, which naturally divides itself into five distinct sections :—

1. The consideration of all hepatic derangements attended with a yellow discoloration of the skin, due to a temporary or a permanent suppression of the biliary secretion.

2. To the consideration of hepatic diseases equally associated with yellow skin ; but in which the discoloration is due not to a suppression of secretion, but to an obstruction to the biliary excretion.

3. To the consideration of hepatic affections with which a yellow skin is neither necessarily nor even so much as usually associated.

4. To the consideration of hepatic ascites.

5. To the diagnosis, pathology, and treatment of diseases of the gall-bladder.

Finishing up with a special chapter on hints in differential diagnosis, and prognosis.

CHAPTER VI.

BILIOUSNESS.

The first disorder of the liver which merits special attention is that very common form of complaint known to all, either by personal experience or social repute, under the vernacular title of 'biliousness,' a 'bilious attack,' a 'sick headache,' a 'torpid or sluggish liver,' all of which are terms applied by laymen to what they suppose are symptoms due to one and the same pathological condition. While the title of 'functional hepatic derangement' is by medical men frequently employed to denote the disordered conditions of the liver usually included in the vernacular generic names of biliousness, &c., under the equally erroneous supposition not only that functional hepatic derangement is *per se* a morbid state, but that all hepatic functional derangements arise from the same pathological conditions of the organ.

I shall now proceed to point out the errors on both sides, and try and rescue from longer misrepresentation the three perfectly distinct pathological

conditions upon which the chain of symptoms commonly denominated biliousness depends.

Although biliousness is generally considered a trivial ailment, it is yet so common—notwithstanding its being an easily preventible one—that I think it is worth while for me to consider separately, though concisely, each of the three pathological conditions giving rise to it, which, for the sake of perspicuity, I define as follows :—

A. Acute biliousness—due to a hyper-secretion of bile.

B. Subacute biliousness—due to a diminished secretion of bile.

C. Chronic biliousness—due to imperfect secretion, both as regards the quantity and the quality of the bile.

To this latter form alone can with any show of reason be applied the terms torpid or sluggish liver.

The Etiology, Symptoms, and Pathology of Biliousness.

The fundamental cause of all the three forms of biliousness is, most undoubtedly, hereditary predisposition to hepatic derangements. When, however, the predisposition exists, there are not wanting subsidiary circumstances which readily act as proximate or immediate exciting causes of a bilious attack. Among the most prominent and the easiest traceable are errors in diet, deficient muscular exercise, hot weather, and habitual constipation.

The intrinsic value, as well as the *modus operandi*, of the first four of these factors in producing hepatic derangements has already been sufficiently pointed out at page 241, so that it only remains for me to explain the *rationale* of the effects of constipation in the production of biliousness.

The reason why constipation is a fruitful source of hepatic derangement is, I think, easily accounted for, and is this. As already pointed out at page 161, the more active the peristaltic action of the bowels, the greater is the effect of the stimulating reflex action exerted upon the contractile muscular coats of the bile-ducts and gall-bladder, and the more powerful the amount of their contraction, the greater is the freedom with which the *se*creted bile is *ex*creted by them into the intestines. Hence it is, as was pointed out under the head of the *rationale* of the therapeutical action of purgatives in cases of jaundice, that brisk action of the bowels favours the expulsion of bilious stools, and *vice versâ*. The more sluggish the action of the intestines is, the less reflex stimulus is conveyed from them to the gall-bladder, and consequently the smaller is the amount of bile expelled therefrom. Moreover, sluggish action of the bowels retards the portal circulation, and induces a congested state of the liver highly favourable to the accumulation of bile in the ducts and gall-bladder, and therefore in that way it still further indirectly con-

duces to biliousness. It is an almost equally common thing to hear persons speak of torpid bowels, as of torpid liver ; and no wonder, seeing that the one condition is so very frequently the concomitant, if not even the exciting cause, of the other. Torpid bowels leading in turn to constipation, flatulency, furred tongue, headache, languor, and biliousness.

After what was said in the chapter upon the different weights of the human liver at different periods of life, and the remarkable manner in which the size of the organ gradually diminishes in relative proportion to the weight of the body as age advances (page 42) and its functions become less and less essential in the animal economy, one cannot feel surprised that the disordered condition which we call biliousness should in like manner vary in proportionate frequency as age advances. Or that the three forms of acute. subacute, and chronic, in which it appears, should be relatively more commonly met with at one age than another. Thus, for example, although all the different forms of biliousness may occur at any time of life, each form seems to have a particular predilection for particular ages. The first or acute form—that from hyper-secretion of bile —is most common in childhood. The second or subacute form, arising from diminished biliary secretion, is most frequently met with in youth. The third or chronic form, arising as it does from sluggish

biliary secretion, is in general only met with in adults, more especially those who have been for some years resident in hot climates. Moreover I think it may be safely said that, as a rule, the severity as well as the frequency of all kinds of bilious attacks, in predisposed individuals, gradually diminishes after puberty ; that the first two forms are rarely if ever met with in persons over forty years of age ; and that the acute form is decidedly most common among children and young women, especially among brunettes at the period of puberty, who in general speak of these attacks as 'sick headaches.'

So much for the exciting causes of biliousness in the predisposed. I say predisposed, for unless a predisposition, either hereditary or acquired, actually exists, neither over-feeding, deficient exercise, hot weather, nor habitual constipation, are of themselves sufficiently powerful exciting agents to induce a bilious attack. I now come to the symptoms.

Symptoms presented by the three forms of biliousness differ considerably from each other. In the acute form alone there is either bilious vomiting or diarrhœa. In the subacute, headache is usually the most prominent symptom. While the chronic attack is chiefly characterised by the sallowness of the complexion and the 'good-for-nothingness' of the patient's feelings.

The attack of the first usually terminates within

forty-eight hours. Of the second, within four days. While that of the last may extend over months.

The other respects in which the three forms of bilious attacks differ will be pointed out when each is being separately considered ; but in the meantime I shall give a general summary of the symptoms which are popularly supposed to characterise an attack, which everyone imagines he can easily diagnose from his personal familiarity with it, in consequence of scarcely a single dark-complexioned person in this country, in childhood and early youth, ever escaping a hot summer without suffering from at least one more or less severe bilious attack.

The general symptoms are usually ushered in either by bilious vomiting or a frontal headache. The form of headache is in general very significant, as it is usually situated immediately above the eyes, the upper half of the balls of which are painful on pressure. Sometimes the headache is more or less parietal, and occasionally, though more rarely still, occipital.

Where there is no marked headache, drowsiness is a frequent accompaniment of a bilious attack. While, on the other hand, a condition of sleeplessness is almost as common when the headache is severe.

A bilious headache is frequently associated with intolerance of light, in some instances almost amounting

to photophobia. In most cases, too, there is dimness of vision, almost amounting to a partial blindness; for the sufferer may be unable to read ordinary type at the usual distance, on account of the words running, as it were, into each other. What are technically called muscæ volitantes—black, fly-like specks before the eyes—are common.

Giddiness and great lassitude are likewise symptoms often complained of; with loss of appetite, and a bad taste in the mouth. At the same time the skin assumes a decidedly sallow tint, varying in intensity from a mere sallowness up to a yellowness sufficiently *prononcé* to merit the title of 'a slight jaundice.'

The subacute and chronic forms of biliousness are indeed, when their pathology is properly considered, nothing more or less than forms of jaundice from suppression, merely differing from true jaundice as regards degree, and only escaping the title of jaundice from the skin not assuming a sufficiently intense yellow hue.

Such being a brief summary of the general symptoms of biliousness, I now turn to the special characteristic of each of the three forms of the disease.

As regards the first, or, as I call it, the acute, form of biliousness, which arises from a hyper-secretion of bile, which is common among children and young women, it may, as rule, be easily traced to

some error in diet in a predisposed subject—sometimes both slight and temporary. A surfeit of plum-pudding is, as everyone knows, a fruitful source of biliousness among children at Christmas time. And it may occur in the adult from an equally transient cause. In a woman from eating a cheese-cake, or in a man it may (as happened in the case of the old rich bachelor I saw in consultation along with Dr. Macaldin) be traced, as said at page 253, to an injudicious dinner, which, in the case referred to, consisted of a pound of salmon steak and a bottle of champagne. This form of acute bilious attack is sudden in its onset, rapid in its course, and temporary in its duration. The principal and most characteristic of its symptoms being bilious vomiting, with or without diarrhœa. There is but little sallowness of the complexion, no light-coloured stools, and although the urine is usually scanty and high-coloured it contains —at first at least—no abnormal quantity of bile-pigment. The tongue is foul, but the pulse is little affected. Sometimes even slower than natural, from considerable nervous prostration accompanying the attack.

In acute biliousness, headache is not a prominent symptom, except in the case of weakly-nerved people; nor, indeed, are any other cerebral symptoms, as a rule, present except the depression just alluded to. Indeed, I have more than once been

told that even the headache was nothing until after the sickness commenced. Which fact leads me to suppose that the headache in these cases is occasionally not due to bile-poisoning at all, but merely to the pain caused by an increased flow of blood to a hyper-sensitive brain substance during the violent and repeated efforts of vomiting ; and that may probably account for the fact that young ladies have christened these forms of attack 'sick headaches.' Very often, indeed, sudden sickness seems to usher in the attack. For the patient, after feeling a little out of sorts, perhaps for a few hours only, all at once complains of being dreadfully sick, rushes to a basin, and almost immediately empties the stomach. Then, after repeated retchings, brings up a quantity of pure yellow-greenish, sometimes even bluish, bile, recognisable both by its taste to the patient, and by its look to the doctor. If the latter has any doubt about the nature of the coloured liquid, he can always easily satisfy himself on this point by adding to the vomited matter a few drops of strong nitric acid, when the intense play of colours produced by it on the bile-pigment will at once yield evidence of its presence.

The Pathology of acute biliousness appears to me, on physiological data, simple enough. As I said in my definition of this form of the disease, the symptoms are due to a hyper-secretion of bile. The

mechanism of which is, I believe, as follows :—A temporary hepatic capillary congestion is suddenly induced in a person predisposed to it by an injudicious meal of indigestible food and drink. Or by a mere over-indulgence, during hot weather, in a single rich stimulating fatty or saccharine dish at a time when insufficient bodily exercise is taken to burn off the excess of hydrocarbons in the blood, as explained at p. 65. The introduction into the portal circulation of the elements of food abnormal either in quantity or quality induces congestion of the hepatic capillaries in precisely the same way—although, of course, in a less exaggerated form—as occurs when stimulating substances, such as ammonia, alcohol, ether, and chloroform, are artificially introduced into the portal vein.[1] In consequence of this temporary capillary congestion more blood than normal is carried to the hepatic secreting cells, and as a natural consequence their action is accelerated, and, their function being to secrete bile, an excessive secretion of bile is the result. More bile being secreted than the system requires, the gall-bladder gets over-filled with it, and at length, probably owing to some merely trifling cause, suddenly expels its surcharged contents into the duodenum, and the duodenum, rebelling against the

[1] See author's experiments on the production of diabetes artificially in animals by introducing stimulants into the portal circulation, already referred to at page 61.

intrusion of such a large amount of irritating bile, makes violent peristaltic efforts to expel it. This excessive peristaltic action is communicated to the stomach; the patient feels sick, is attacked with violent retchings, and after repeated straining efforts succeeds in vomiting up probably nearly the whole of the bile which was thrown into the duodenum. If not, the remainder soon finds its way into the ileum, and, by there exerting its normal physiological purgative effect, brings on a bilious diarrhœa.

This acute form of bilious attack may here end, and oftentimes does here end, almost as suddenly as it commenced, without any treatment whatever being necessary. From Nature herself having got rid of the whole of the offending bile that was thrown into the duodenum, the symptoms its presence there induced speedily disappear, and in a couple of days or so the patient is himself again.

Not so, however, with the subacute bilious attack, depending on the diminished secretion of bile, which I have now to consider. In the first place, the immediate or proximate exciting cause of SUBACUTE BILIOUSNESS is not one or two accidental indiscretions in diet, but a continued habitual indulgence in richly oleaginous and saccharine foods. Coupled, it may probably be, with an excessive consumption of fermented liquors. Every patient liable to subacute biliousness, however, is not to be put down either as

a glutton or a drunkard. For an hereditarily or constitutionally predisposed individual, in hot weather, and from taking little exercise, will often be attacked with the subacute form of biliousness, though exceedingly moderate both in food and in drink. And to me the explanation of this is simple on the supposition that the subacute form of biliousness is the direct effect of a subacute form of chronic hepatic capillary congestion (see p. 100). In fact being merely a minor form of jaundice from suppression, though, strange to say, it springs from the identical same pathological cause as that which produces the previously considered acute form of biliousness—biliary hyper-secretion. This reads very like a paradox, and as such it might be regarded. Yet though seemingly an absurd proposition, it is nevertheless a true pathological fact, easily enough understood when the explanation of it is given. Its explanation is :—

The first stage of hepatic capillary congestion leading to a hyper-secretion of bile, by being prolonged in its action, inevitably leads to a *second stage of Diminished Biliary Secretion.* Which is brought about thus :—The hepatic capillaries not only being themselves engorged, but the bile-ducts after a time becoming also cram-full of secreted but not excreted bile, combine together to exert a sufficient amount of backward pressure on the secreting hepatic cells to effectually prevent them performing their work pro-

perly. Hence the normal bile ceases to be secreted in its usual quantity. The colouring matter and other biliary ingredients which the liver removes from the blood remain behind in the circulation, and, gradually accumulating there, give rise to the chain of symptoms usually included in the generic name of a bilious attack. To wit, a sallow complexion—from the serum of the blood becoming overloaded with the non-eliminated bile-pigment, and exuding into and staining the pigmentary layer of the cuticular *rete mucosum*—headache, giddiness, disordered vision, sickness, and prostration. This latter series of symptoms, again, are the result of the toxic action of the biliary substances circulating in the blood on the nervous system. In fact this second form of biliousness is nothing more or less than a slight or incipient stage of jaundice. Which it only escapes from being called in consequence of the amount of bile-pigment effused into the skin not being sufficient to stain it distinctly yellow, nor the suppression of the biliary function being sufficiently complete to entirely exclude bile from the stools and cause them to assume a distinct pipeclay colour. There is clearly, however, a sufficiency of the elements of the bile retained in the blood to produce slight symptoms of bile-poisoning. Not the serious ones of delirium, convulsions, or coma, but the minor ones of giddiness, confusion of ideas—or perhaps it would be better to say

loss of clear-headedness—dimness of vision, and intolerance to light, sound, or contradiction. It is no uncommon thing, we know, to hear it said of an angry man that 'his bile is up.' And it is almost equally common to hear that a person takes a jaundiced view of things. In fact, popular ideas, I think, are much oftener correct ideas than most people suppose. When I was a boy, hypochondriacism was a favourite word in everybody's mouth to define what Webster in his popular dictionary calls dyspeptic melancholy. Now, if we look at the etymology of the word, it at once leads us to the liver—the organ occupying the hypochondriac region. The mind and the liver are marvellously connected. Who amongst us does not know that a dose of blue pill will dispel an attack of the 'blues'? How depression of spirits, irritability of temper, and peevishness vanish after a good clearing out of the bowels by a dose of calomel? How the discontented melancholic man is at once himself again, a pleasure to his friends and a comfort to himself, after a copious bilious motion? Do not our 'alienists' give the name of melancholia to a form of monomania frequently associated with derangement of the hepatic functions? So that in very many instances the way to cure the mind is to treat the body. As soon as the liver's functions return to their normal state, groundless fears, melancholy forebodings, ima-

ginary anxieties, one by one dwindle into shadows, become fainter and fainter, and finally entirely disappear.

Fortunately we have it in our power to make a rapid and safe cure of these cases, and this, I think, must be self-evident after what has been said regarding the pathology of subacute biliousness. Nor can it fail to be readily understood how a smart dose of a mercurial should be the sheet-anchor of our treatment. In fact, a single dose of calomel, say from three to five grains, given to an adult on the verge of a subacute bilious attack will dispel it as if by magic, and, even after it has commenced, will cure it, in the majority of instances, within twenty-four hours after the time of administration.

Podophyllin, and all the other forms of hepatic stimulating purgatives, act well enough in slight cases ; but mercury, either in the form of calomel, blue pill, or grey powder, is the king of remedies when once a subacute bilious attack has fairly set in. With or without a purgative superadded, according to the necessities of the case. (For the *rationale* of its action, see the article on Mercury in the chapter on the General Therapeutics of Hepatic Disease, p. 156.)

I now come to the special consideration of the third or chronic form of biliousness, the one arising from a sluggish or torpid condition of the liver.

This condition, as was before said, is most commonly met with in adults who for a time have been resident in hot climates, where the causes producing a state of chronic congestion of the hepatic capillaries may be said to be in the ascendant (see page 241). The secretion of bile, like the majority of other glandular secretions, not only varies normally in quantity and quality at different periods of the day, but is also liable to special variations according to the quantities and qualities of the food taken, even by the most healthy, as well as according to the stage of digestion. Besides these normal variations in the rapidity of the biliary secretion depending upon physiological causes, there are other abnormal fluctuations in the total amount of bile secreted within a given time, due to purely pathological causes. At page 73, I not only pointed out the effect of nerve influence in diminishing and even entirely arresting the biliary secretion, but also gave a striking example of the effect of inflammation in totally arresting glandular secretion. It is easy, therefore, to understand how a chronic state of hepatic capillary congestion may give rise to an equally chronic condition of diminished biliary secretion. For, as already shown, while the primary effect of moderate capillary congestion is to accelerate, the secondary effect is to diminish glandular secretion. No organ whatever, indeed, with its capillaries in a

chronically congested state, ever pours out its secretion in normal quantity or of normal quality, and the liver is no exception to the rule. Hence a sluggish or torpid liver induces an attack of biliousness —or sub-jaundice as it might be called—from a chronic partial suppression of the biliary function. The symptoms of which are precisely the same as those arising from the second or subacute form. Only differing from them in their being of a more thoroughly chronic character.

Treatment.

The acute bilious vomiting and diarrhœal forms of attack usually cure themselves. For the subacute form, a smart mercurial purgative is all that is in general required ; but the third or chronic form of biliousness is much less under the control of remedies. A single smart dose of a mercurial will not cure it. The chronic congested condition of the capillaries is gradually to be attacked, and here it is not mercury but alkalies—especially alkaline purgatives, such as sulphate of soda and potash—which prove of the most marked service. If the patient chances to have been for some time resident in a hot climate, he must be carefully questioned with the view of ascertaining if the congested state of the liver has been induced by malaria, as will be subsequently shown at page 365 ; for if it has, his cure is not likely to be effected

without the administration of quinine, or a decoction of Chionanthus virginica (see page 182). The course of procedure which I find in ordinary cases successful is as follows :—

First, to give a single smart mercurial purgative. To a strong adult, probably a powder consisting of :—

R	Calomelanos . . .	gr. iij.
	Pulv. Rhei . . .	gr. iv.
	Magnesiæ . . .	gr. xij.
		M. Hora somni sum.

After the free action of this mercurial alkaline purgative, in order to stimulate the secreting function of the liver, and retain the bile in as fluid a state, by getting into it as much taurocholate and glycocholate of soda, as possible, I prescribe half an ounce of the following mixture to be taken, in the intervals of the meals, three times a day in half a tumbler of water.

R	Succi Taraxaci . . .	ʒ xv.	
	Sodæ Sulphatis . . .	ʒ vj.	
	Sodæ Bicarbonatis . .	ʒ ij.	
	Inf. Calumbæ ad . .	℥ vj.	M.

Telling the patient to shake the bottle well before measuring out his doses.

Once the attack has been subdued, the next question is 'How can its recurrence be prevented?' For just as a horse that has once stumbled and fallen is

apt to come down again, so a patient predisposed to biliary derangement, if great care be not taken to prevent it, is almost sure, sooner or later, to be seized with another attack. Fortunately for the reputation of our art, the doctor's advice can here be of great service; for although he cannot eradicate the tendency, he can ward off, and oftentimes even totally prevent, the attacks, by prophylactic treatment, therapeutic and dietetic.

If the reader has paid due attention to what was said in the chapter devoted to the consideration of the functions of the liver, and will take the trouble of carefully balancing it in his mind along with what was said regarding the etiology of biliousness, it will be self-evident to him that the prophylactic treatment must be directed solely to removing the cause; for, as everyone knows, no effect is producible without a pre-existing cause.

The regulation of the patient's food, drink, and exercise, is therefore the first—I might almost say the chief—thing requiring to be considered; for with a well-regulated diet and *régime* attacks of biliousness might be reduced to things almost unknown.

It is not only the quality but the quantity of the food that has to be regulated. For an excess of the most wholesome of foods acts in persons predisposed to biliousness as an exciting cause. Therefore it is good policy never to allow the patient at any

time to eat more than the wants of his system demand. If he be corpulent, even put him on short commons. Stop his beer, and reduce his wine to a couple of glasses of hock or claret a day. If he be thin or only moderately stout, only stop all salted foods, ham, bacon, hung beef, bloaters, Finnan haddocks, &c. Order him to take nothing but fresh foods. Not too fatty, and rather underdone. To avoid shellfish and pastry. To make his chief meal in the middle of the day. To take as much walking exercise as he possibly can without actually fatiguing himself, and to go to bed early.

For further dietetic details see page 239. In some persons with a more than usual tendency to biliousness, traceable to sluggish biliary secretion, besides chronic hepatic capillary congestion there sometimes exists also defective nerve action. The secretory nerve twigs appear as if they had literally as well as figuratively gone to sleep, and I believe it is by waking them up, as it were, with small doses of nux vomica, that homœopathic practitioners have obtained the credit of being able to cure bad bilious attacks. Certainly, for some years past, I have taken a leaf out of their book, and treated many cases of torpid liver with strychnia with marked benefit. The rationale of the action of the strychnia in these cases appears to me to be precisely the same that it is

in mild cases of paralysis, where it evidently calls nerve action into play, as is visibly seen by the paralysed muscles twitching under its influence. When given in torpid liver from defective nerve influence, the stimulating effect of the strychnia upon the nerves is, I think, rendered equally patent to our minds, though not to our eyes, by the increase of the biliary secretion.

Moreover, as a sluggish state of the bowels is a usual concomitant of a sluggish biliary secretion, I usually combine the strychnia with belladonna, which has a specific action on the intestines, and produces easy motions without purging the patient. In order to gain a still further advantage, I give, as a rule, the strychnia and belladonna in combination with taraxacum, generally in the form of a dinner pill to be taken at the commencement of the meal ; and as I should like others to try my plan, its formula is as follows :—

℞	Strychniæ Acetatis . . .	gr. j.
	Ext. Belladonnæ . . .	gr. vj.
	Aloes Socotrinæ . . .	gr. xiv.
	Ext. Taraxaci . . .	ʒ ij.

M. Divide in pil. xxxvj. ; obduce argento et signa. Sumat unam diebus singulis.

From what has now been said it is evident that all the three forms of biliousness are greatly under

the control of medicine, diet, and *régime*. With careful prophylactic treatment, biliousness might be made a thing unknown. For even after the premonitory symptoms of an acute or subacute attack have manifested themselves, it is almost invariably possible to nip it in the bud by a single brisk mercurial alkaline cathartic dose of medicine. And, as prevention is invariably better than cure, the opportunity of arresting an attack ought never to be allowed to slip.

I have now only further to add that the foregoing prescriptions ought to be regarded in the light of mere samples ; for, as I said before, I do not pretend to be able to lay down definite lines of treatment in individual cases. For, in prescribing, constitutions, as well as idiosyncrasies, require to be considered quite as much as the special disease under consideration. Consequently it is general principles which I can alone indicate, and merely as such are my remarks to be considered.

CHAPTER VII.

INTRA-UTERINE, CONGENITAL, AND HEREDITARY JAUNDICE.

Nor only are infants, from the very moment of their birth, liable like adults to be attacked with a variety of forms of jaundice, the direct result of different kinds of liver derangement, but, while yet even within their mother's womb, they may suffer from the same affection, and in identically the same forms. Namely, jaundice from suppression and jaundice from obstruction. This fact can surprise no one who is aware that the secretion of bile, like the secretion of urine, begins long before birth—begins, indeed, so soon as the secreting cells of the fœtal liver are formed [1]—and hence it is that children have again and again been born labouring under an attack of well-marked jaundice. Although both malarial and idiopathic hepatitis are frequent causes of intra-uterine jaundice, the most common cause of all is

[1] In proof of this statement that the biliary function begins as soon as the hepatic tissue is formed, I may cite the observation of Zweifel who found not only bile pigment, but bile acids in the contents of the intestines of a three months fœtus. (*Centralblatt*, No. 59. 1874.)

congenital malformation, and imperfection of the bile-ducts. Having early in my professional career, even before I graduated—while acting as house surgeon to the Edinburgh Royal Maternity Hospital—met with an exceedingly well-marked case of this kind, I have ever since given considerable attention to the literature of all the various forms of infantile jaundice; and I deem the subject of sufficient importance to go pretty fully into it.

To begin with, I shall consider the spurious form of jaundice which, from ignorance of its true pathology, received the incorrect title of 'Icterus Neonatorum.' While, strange to say, in the morbid state to which this learnedly sounding name is given, there does not exist one single sign or symptom of jaundice whatever, except it be the slightly yellowish tint of the skin. No high- or saffron-coloured urine is present. No pipeclay-coloured stools. Nothing whatever that can be traced to a derangement of the biliary function, any more than can be the equally sallow complexion of the cuticle met with in cases of chlorosis, syphilis, or other forms of blood-poisoning. Indeed, be the cause or causes of the so-called icterus neonatorum what they may, among the chief of them must at least be reckoned a defective oxygenation of the red hæmatin of the blood, just as occurs in the true chlorosis of the male as well as female human adult. In icterus neonatorum the skin is never yellow at the moment

of birth as in true jaundice. On the contrary, it is of the rosy tint peculiar to the new-born babe, and it is not until twenty-four, often indeed not until seventy, hours after birth that it assumes a yellow tint. The rosy hue first changes to a dirty white, then to a dull pale yellow. Exactly like that of the skin in cases of true chlorosis in the adult. At the same time be it observed that the health of the child is not impaired beyond what it was before the yellowness appeared.

This form of affection—which, on account of its pathology, I shall henceforth name Chlorosis Neonatorum—is only seen in weakly children chiefly, among the immature, and is, I believe, as before said, entirely due to the imperfect oxidation of the blood from the defective respiratory powers of the child, associated, it may be, with a vitiated atmosphere. Which still further tends to increase the imperfect oxidation of the hæmatin. Especially when it is combined with the depressing influences of external cold on the low vital powers of an immature or weakly infant.

This idea of its etiology is very strongly supported by the fact that in maternity hospitals, where attention is paid both to ventilation and temperature, chlorosis neonatorum is an exceedingly rare disease. While, on the other hand, in institutions in which sanitary arrangements are defective, as in many—I

might even say, from personal observation, in most— of the Continental lying-in charities, this so-called icterus neonatorum is of frequent occurrence. It is easily differentiated from true congenital jaundice by the fact that although the skin of the child is yellow, neither are the conjunctivæ of a yellow hue, nor the urine of a saffron tint, nor the stools of a pipeclay colour. Facts quite sufficient of themselves to upset the idea that chlorosis neonatorum is due to a derangement of the hepatic biliary functions, either in the form of a suppression to its secretion or an obstruction to its excretion.

An easy, and at the same time a crucial, way of making a differential diagnosis between cases of chlorosis neonatorum and congenital jaundice, is simply to gird up the child's loins with a clean piece of white linen, and watch the effect the urine has upon it. If the child's urine stains the linen of a yellow tint, the case is one of true jaundice ; if, on the contrary, it merely gives it the ordinary urine stain, the case is as decidedly simply one of chlorosis neonatorum.

Of intra-uterine and consequently congenital jaundice there are almost as many varieties as in the adult, for the simple reason that almost all of the same causes which produce jaundice in the adult are equally potent enough to produce it in the fœtus. Indeed, there are even extra causes for its production

in the unborn babe—congenital anatomical malformation from defective development, one of the commonest forms of which is the entire absence or imperviousness of the common bile duct. As the example of this kind which fell under my notice while I was acting as house surgeon to the Edinburgh Royal Maternity Hospital is well worth putting on record, I shall give it briefly.

A married woman, already the mother of several children, was safely and easily delivered of a male child, whose skin at the moment of its birth was of a well-marked jaundiced tint. Immediately on escaping from the maternal passages, the moment it began to cry, and before it was even separated from the placenta, it passed in a full stream a quantity of dark saffron-coloured urine, which stained the bed-sheets of a bright lemon-yellow hue. The urine had a strong odour, as I know, for I smelt some that went upon my hands. The child seemed quite healthy, though not robust. It weighed 7 lbs. It took the breast, and nothing was thought the least remarkable about it, except that it had a yellow skin. It was however found dead by its mother's side on the fourth morning after its birth. Although the mother, from being a married woman, was not suspected of foul play, I considered it my duty to make a *post-mortem* examination, in order, if possible, to discover the cause of the child's death. At the

necropsy the gall-bladder was found distended to the size of a small hen's egg. The hepatic tissue was stained green, but not deeply. The contents of the intestines were of a pale creamy hue. The urine was dark in colour. Indeed, there was present every pathological sign usually met with in cases of jaundice from obstruction. A careful search was made for the cause of the disease, and it was soon discovered that the common bile-duct was impervious; in fact, its lower part looked like a mere cord of solid fibrous tissue; whereas both the hepatic and cystic ducts were enlarged, and full of fluid bile, just like that in the distended gall-bladder.

As many forms of liver disease, and consequently jaundice, are frequently hereditary, it will astonish no one when I say that a jaundiced mother may give birth to a jaundiced child. Dr. Moxon met with a remarkable example of this kind.

The case was that of a man, aged 30, who said he was born yellow, and had remained yellow ever since. His brother was also born jaundiced, and they were both supposed to inherit the disease from their mother, who had jaundice and died jaundiced at the age of 54. Dr. Moxon's patient, with the exception of the jaundice, was otherwise in good health. Not only his skin, but his conjunctivæ were yellow. His urine contained bile-pigment, and deposited lithates. He felt giddy when looking up, as

many bilious patients do. His liver, Dr. Murchison (to whom Dr. Moxon showed the case) says at p. 426 of the second edition of his book on diseases of the liver, was enlarged; but as the measurement given is $4\frac{3}{4}$ inches in the r. n. l., it ought not, properly speaking, to be called an enlarged liver; for many perfectly healthy men have livers of that size. Both brothers had several children, all of whom are reported to have 'become deeply jaundiced two days after birth, the colour of eyes, body, and whole frame being of a deep yellow hue, but disappearing after about a month.'

So long ago as in 1752, Mr. Benj. Cooke reported a case of jaundice in a newborn child, in the 'Philosophical Transactions' (p. 207), which he thought was directly transmitted to it by the male parent through the spermatozoa. The case is so curious that I shall give it *in extenso* in the author's own words.

'A man of about 22 years married a healthy woman much of the same age. Soon afterwards he went to America, and at the end of seven years returned, cachectic, anasarcous, and deeply tinged with the jaundice endemical in hot latitudes. In a few months after his return his wife became pregnant (with her first child), of which she was delivered in due time. The child was born with a jaundice upon it, and died about six months after, under ascitical

and icterical symptoms, of which the mother had not the least impression. Soon after this (and before the husband, though much better, was quite cured) she became again with child, and after about three months' pregnancy turned yellow, and was the whole time of her going with child, and some months after her delivery, deeply affected with jaundice. But the child was born quite fair, white, and healthy, without anything of that distemper on it; and is still living, and the last born.'

These last cases of true intra-uterine jaundice ought perhaps, in the present state of our knowledge, to be regarded as cases of jaundice from suppression, due to some as yet unknown hereditary cause.

More easily understood, but still almost equally curious, is the fact quoted by West in his 'Diseases of Infancy,' that a lady lost three children (out of five) in succession from most intense jaundice; and, although in one instance alone was it accurately ascertained to be due to defective biliary ducts, it is highly probable that, in obedience to the laws of hereditary malformations, the same cause existed in the other two. Dr. West also says that he saw another woman's child die of jaundice from impervious biliary ducts, who had already lost three infants from the same cause. While, strange to say, as illustrating still more strongly the influence of the hereditary transmission of liver disease with jaundice,

her sister's only child died under exactly similar circumstances.

A great number of writers on infantile diseases have stated that hæmorrhage from the navel is almost an invariable accompaniment of infantile jaundice, and most assuredly it would appear not to be rare; for a German physician of the name of Grandidier, published in 1871 a monograph on the subject, in which he makes reference to eighty cases that he found recorded in various journals.

It would appear, therefore, to be an undeniable fact that there actually exists some sort of close connection between the occurrence of umbilical hæmorrhage and infantile jaundice. This connection, however, is perhaps due, as I believe, to the simple fact that infantile jaundice usually occurs in immature and defectively developed children. The umbilical tissues and organs of whom, even when not actually malformed, are at least more feeble than they ought to be. Not only in cases of jaundice from obstruction, arising from imperfectly developed bile-ducts, but likewise in the cases that I call infantile chlorosis, which, as already said, only occurs in children of an abnormally weak state of body. A condition of course very likely to conduce to the accidental occurrence of umbilical hæmorrhage while the ligatured end of the umbilical cord is sloughing away. Which is said to be the exact period of the occurrence of

infantile umbilical hæmorrhage. The sloughing of the end of the cord—barring the hæmorrhage—being in itself, of course, a perfectly normal process. With all due deference to Dr. West, I therefore beg to differ from him *in toto* when he says at page 622 of his sixth edition, 'The bleeding is dependent on a congenital malformation of the hepatic ducts.' For I believe—and I think with good reason, too—that the umbilical hæmorrhage in cases of congenital malformation of the bile-ducts is not due to any deficiency in their organisation, but to a similar species of defective organisation existing in the UMBILICAL BLOOD-VESSELS and navel themselves. In a word, to general deficient developmental power in the constitution of the infant, giving rise at one and the same time to an arrest of development in the hepatic ducts, as well as to an arrest of development in the vessels of the umbilical cord. All these parts are so intimately connected together in fœtal life, by means of the ductus venosus and umbilical vein, that we can readily understand how a deficiency in the ducts may also be associated with a defective development in the coats of the umbilical vessels. Sufficient, at least, to admit of hæmorrhage occurring from them before their divided ends have become perfectly closed up. This theory or view of the matter is strongly supported by the fact that in most of the cases of umbilical hæmorrhage in which the ana-

tomical conditions have been carefully recorded allusion is made to the divided ends of the fœtal vessels having been found imperfectly healed up. While, had they been normal, they ought to have been healed at least by the end of the fourth or fifth day after birth.

Cases of intra-uterine jaundice from obstruction to the flow of bile into the intestines will have again to be referred to further on, while speaking of the various kinds of obstructive jaundice.

Treatment.

I must begin my remarks on the treatment of congenital jaundice by saying a few words regarding the treatment of the spurious kind, which, in order to make its title accord with its pathology, I have changed the name of to 'chlorosis neonatorum.' From having said that it is a morbid state met with in immature and weakly children, it can be readily understood why not medicine but good mother's milk, external heat, and plenty of fresh dry warm air must be the essentials of treatment. When I have said this, I have said all that is necessary to be said upon the subject. So now I turn to the consideration of the treatment of true congenital jaundice. For the one form of which we can do much; for the other, nothing at all.

The treatment of congenital jaundice from sup-

pression of the biliary function is in most cases a very simple affair. A single dose of a mercurial being, in general, sufficient to dispel it. But here I have an important word of advice to give, and that is, to advise my readers *not* to follow the plan recommended in books of giving either the grey powder or the calomel to the baby ; on the contrary, give the medicine to its wet-nurse—whoever she may be—for a dose of mercurial given to the woman who suckles is sure to affect the child who sucks her. Ay, and that, too, much more naturally and satisfactorily than if the medicine be poured down the infant's own throat. There is no fear of the mercurial doing the mother harm, even if it be given within seventy-two hours after the confinement, as I well know from personal experience, or I should not thus unhesitatingly recommend it.

I need say little about treatment in cases of jaundice from a congenital deficiency of the bile-ducts, for, of course, they must inevitably, sooner or later, end fatally.

One might expect even, after what was said in the physiological chapter on how very essential to life is the presence of bile in the digestive process, that it would be almost an impossibility for a child to survive beyond a few days, or at the very utmost beyond a few weeks, with an entire absence of any canal by which the bile could obtain access to the duodenum.

Yet, strange to say, Dr. Nunneley has put on record the extraordinary case of a boy who was born jaundiced in consequence of a malformation of the bile-duct preventing bile from reaching the intestines, and actually lived for nearly seven months. At the time of his death he was deeply jaundiced and emaciated. Emaciated, naturally enough, he would be, from no bile having reached the chyme to prepare and fit it for the process of intestinal absorption and assimilation.

Even in the apparently most hopeless cases, as it is just possible that an error may be made in the diagnosis—for infantile affections of this kind are not easily differentiated—it is always advisable in the first instance to try the effect of a mercurial, given, as above said, through the instrumentality of the wet-nurse.

I now come to the special consideration of the hepatic diseases which give rise to

Jaundice, the Result of Suppression of the Biliary Secretion.

Pathologically considered, they divide themselves into three perfectly distinct classes :—

A. Those arising from Enervation.
B. „ „ „ Disordered Hepatic Circulation.
C. „ „ „ Absence of Secreting Substance.

Although there can be no misunderstanding the meaning of the term 'Jaundice from Suppression,' there may, nevertheless, be some difficulty in comprehending how the skin becomes yellow, and the urine high-coloured, when the secretion of bile is arrested, by those of my readers who have not paid attention to what was said regarding the physiology of the biliary secretion (p. 100); it may therefore be as well for me to remind them that while the liver's function is to manufacture certain biliary constituents out of the elements of the blood, its duty is merely to extract therefrom certain others which exist therein preformed. Hence it is perfectly evident that when the secretion of bile is arrested, those substances alone which the liver itself manufactures are wanting. While those again which it merely extracts preformed from the blood remain behind and accumulate in the circulation. Just in the same way as urea accumulates in the circulation when the urinary secretion is in a like manner arrested.

Hence, when the biliary secretion is arrested, biliverdin—which is one of the substances which exist in the blood, and which the liver only extracts from it in a preformed state—accumulates there until the serum gets completely saturated with it, and assumes a dark saffron tint.

The skin and kidneys, from their assuming vicariously the function of the liver and eliminating the

biliverdin, have their secretions impregnated with it—the sweat becomes yellow, and the urine of a saffron hue. At the same time the cells of the cuticular rete mucosum being unable to eliminate all the pigment brought to them for excretion become filled with it, and the discoloration of the skin, which is termed jaundice, is the direct consequence thereof.

The order of occurrence of these changes is that, on the second day of arrested biliary secretion, the urine becomes high-coloured ; a day or two later the skin and the sweat begin to assume a yellow tint. While in severe cases, within a week or two, the milk, the tears, and the sputa, as well as the serum in the thoracic and abdominal cavities, become of a more or less decided yellow hue.

From this it is seen that I regard the production of the yellow skin and high-coloured urine of jaundice as being simply due to the non-excretion by the liver of biliverdin from the blood, quite independent of the presence or absence of the other constituents of the bile. The effects produced by which will be referred to elsewhere. Meanwhile I shall now proceed to consider separately the further pathology of the three subdivisions of jaundice arising from suppression, as given above.

CHAPTER VIII.

JAUNDICE AS A RESULT OF ENERVATION.

It is now a well-established physiological fact that all secretions are under the direct influence of the nervous system. Stimulate a nerve supplying a gland, and its secretion is accelerated. Stop nervous action by dividing the nerve, and secretion is instantaneously arrested. Again, just in the same way as volition can produce or suspend muscular movement, mental influence can hasten or retard glandular secretion. As an illustration of this fact, I need only call to mind the influence the mere sight of savoury food has in exciting in a hungry man the salivary secretion, and the effect of bad news in arresting it. Exactly the same influence as is here alluded to is exerted by the mind over the biliary function. If, for example, as Bernard first observed, a dog with a biliary fistula is caressed, the secretion of bile is actively continued ; if, on the other hand, the animal is suddenly ill-used, the secretion of bile is instantly arrested. While if he is again caressed, the secre-

tion is re-established, and the bile flows drop by drop from the end of the canula.[1] Here the influence on the biliary secretion is entirely produced through the intervention of the nervous system; and if such effects as are here described occur in the dog, we can surely have little difficulty in understanding how the biliary secretion may equally be influenced in the highly-developed organisation of the human being. Indeed, every one must have felt how quickly sad tidings received during a meal not only destroy the appetite and retard digestion, but occasionally alter the complexion. This effect, that all of us must have experienced in a slight degree in our own persons, several may have observed to a greater extent in the persons of others, even to the production of well-marked jaundice. At this very time (this was written in 1861) I have under my care a young married lady, who during the last two years has twice suffered from an attack of jaundice induced by witnessing her child in convulsions, and this I regard as an example of jaundice from enervation.

In the 'Dictionnaire des Sciences Médicales,' 1818, p. 420, two very remarkable examples of sudden jaundice arising from mental shock are recorded. The first is that of a young soldier who, on being prevented from wreaking his vengeance upon a man who

[1] Look at what has been said regarding the nervous influence on the biliary secretion at p. 102 under the heading 'Etiology of Jaundice.'

had insulted him in public, suddenly became jaundiced, and in a few hours afterwards died in delirium and convulsions. While the second is that of a young priest who became jaundiced, and immediately died, after having been frightened by a mad dog. In the 'British Medical Journal,' again, of November 19, 1870, the following interesting case of jaundice produced by mental anxiety is recorded by Mr. Churton of Erith. I give it in his own words :—

'In October 1868, I attended a married lady, aged 30, for jaundice following mental and physical fatigue. The ordinary remedies were used. The nitro-muriatic acid was of the most service ; but the discoloration persisted for some weeks. Six months after this, she had several visitors staying in the house, and, having little inclination for society, was somewhat disturbed by attending to them, and by the addition to the ordinary cares of her household. In the midst of this anxiety, one of her children, subject to asthma, had a severe attack one evening, and was in considerable distress all night. Next morning, at five o'clock, I found her sitting up in bed, rocking to and fro, and complaining of acute pain in the hepatic and gastric regions. Pulse 72 ; temperature 98·4 degrees. She showed slight but unmistakable symptoms of hysteria —quivering eyelids, &c. Ten grains of the bromide of potassium were given, therefore, every four hours. The first dose cured her of all pain at once. On the fol-

lowing day, however, I found her completely jaundiced, and the urine of a dark brandy colour. The bromide was continued, but less frequently ; and an aperient pill (podophyllin with colocynth and henbane) was given. On the next day, the jaundice was less intense ; and three grains of the bromide with infusion of calumba were given three times daily. Two days afterwards the yellowness had entirely gone, and the urine was of a natural colour.'

Jaundice arising from enervation may be regarded as the most typical form of jaundice from suppression. For there is no organic change in the hepatic tissue. No premonitory obstruction to the natural flow of bile, either into the intestines or the gall-bladder. There is, indeed, no visible cause for the jaundice whatever. The liver simply appears to be 'on strike.' It ceases, and that too occasionally at a moment's notice, to do its accustomed work. Bile is no longer secreted or excreted. Jaundice is instantly developed. The conjunctivæ and skin become yellow, the stools pipeclay-coloured, and the urine of a saffron hue. Cerebral symptoms supervene ; convulsions, delirium, and coma follow. While death frequently ends the scene. All this takes place, and yet not a particle of lesion, not a sign of the cause, is detectible in the body after death. If not the *rationale* of the jaundice, at least the cause of the fatal issue, is left entirely to hypothesis. We see from the jaun-

dice that the colouring matter of the bile has accumulated in the circulation. We guess from the nervous symptoms that bile has poisoned the blood. We imagine that this has been the direct effect of enervation. But further all is doubt and obscurity. Seeing that the skin became yellow and death ensued within the course of a few minutes after the application of the supposed exciting cause. Time, seemingly, having been an element omitted from the calculation. For the patient was well at one moment, jaundiced in the next, and dead in the succeeding. The effect appeared, as it were, to trip up the heels of the cause.

I may mention that in the 'Annuaire de Thérapeutique,' 1846, is reported a case of jaundice from great JOY in a man—a sex not usually supposed to be very emotional—and although doubting the accuracy of the theory, I willingly admit the possibility of the fact, from knowing as I do that, even in matters medical, extremes meet. No more striking example of which can I give than by referring to the fact that extreme cold is as conducive to hydrophobia in dogs as extreme heat. Kane having during his Arctic travels lost sixty dogs from this cause alone. It is just possible, then, that intense joy may act like intense fright in producing jaundice.

The extreme rapidity with which the skin assumes a yellow tint in cases of sudden jaundice has been

hitherto considered to be an unaccountable mystery. To me it appears nothing so very extraordinary, when the physiology of the biliary secretion is properly understood. For, as the colour of the skin depends entirely upon the effusion of the yellow pigment which exists preformed in the blood, it is easy to understand, when, in consequence of great nervous shock, the elimination of the pigment by the excreting cells of the liver is suddenly arrested, it at once and instantaneously begins to be vicariously eliminated from the blood by the skin, the rete mucosum of which immediately, in consequence thereof, assumes a yellow hue.

Moreover, as an aid to the more easily comprehending how enervation may produce jaundice, I may remind the reader that the to us invisible effect of nerve paralysis on the capillaries of the liver is no doubt analogous to, if not even precisely the same as, the, to us, visible action of the paralysis of the nervous system upon the capillaries of the skin of the face and ears of the rabbit, which follows an artificial mechanical interruption to the function of the eighth pair of nerves; as occurs when their nerve-cord is divided in the animal's neck by a scalpel or a pair of scissors. No sooner is the division made than the capillaries of the same side of the face and head become visibly congested and enlarged. This effect is markedly apparent in the ear if the animal

selected for experiment be an albino rabbit. Not only is there congestion, but actual turgescence and increased temperature, of the whole side of the face and head, just as if a pathological and not a mere physiological inflammation had been induced. If this, then, is the visible effect of interrupted nerve action in the eighth pair of cerebral nerves following upon their paralysis by artificial section, surely it requires no great stretch of the imaginative faculties to enable us to believe that it is not only possible, but even highly probable, that a corresponding effect is produced on the invisible capillaries of the brain. Following up this line of argument still further, can we not see in it an explanation of how the enervation of the liver is attended with such an amount of capillary congestion as is sufficient to induce jaundice from suppression? Nay more, may we not go so far (although the ground is no longer so firm under us) as to believe that the enervation which induces jaundice is at the same time potent enough to act in a similar paralysing manner on the nerves of the cerebral capillaries, and thereby, on the congestive hypothesis, furnish us with a clue to the well-recognised and ofttimes observed fact that one of the most characteristic concomitants of jaundice the result of enervation, whether from nervous shock or the introduction of toxic agents into the system, is its invariable, or at least almost invariable, association with cerebral

disturbance? Cerebral congestion being the most potent of all pathological causes of mental disturbance. I am perfectly aware that this theory of mine is not unlikely to be gainsayed, for the *fons et origo* of cerebral symptoms in cases of jaundice is still a matter of virulent dispute among hepatic pathologists. One set believing that they are in all cases due to the accumulation in the blood of those substances which are normally extracted preformed or eliminated from it by the liver, to be manufactured into biliary ingredients. Another set, ignoring this bile-poisoning theory altogether, assert that the derangement in the cerebral functions is merely due to the imperfect elaboration and elimination of the effete matters of the tissues—such, for example, as the urea, the uric acid, the uro-hæmatin, and other normal urinary products —which it is the duty of the kidneys and not of the liver to eliminate. They say, too, that in by far the majority even of cases of contagious jaundice, which are always attended by marked cerebral disorder, the elimination of urea is invariably found to be diminished, and moreover that in these cases Dr. Blair has detected such an excess of carbonate of ammonia both in the expired air and blood of patients, as to lead him to the belief that ammonæmia (from the decomposition of urea in the circulation) may be at least one, if not the sole, cause of the cerebral symptoms.

This last class of theorists, however, only strike at one half of the question. They strike at the cerebral symptoms ; but they leave the jaundice, with which they are associated, untouched. And as it is often the case that truth is found in the centre of two or three differing sets of conflicting opinions, probably the truth might be here found by combining into one great whole the separate theories advanced on either side.

At the same time, it must be in the meanwhile admitted that the rapidity with which not only cerebral symptoms, but an actual jaundiced condition of the skin, supervene upon sudden mental emotion, is of itself a sufficient reason for adopting, for the present at least, my theory of the congestive action of enervation on the hepatic and cerebral capillaries in preference to any of the others.

Mental emotion, especially of a sudden and disagreeable kind, is not only capable of producing jaundice from arresting the biliary secretion, but may actually cause very serious hepatic structural change. Such, for example, as occurs when acute atrophy of the parenchyma of the liver follows intense fright. Mental emotion, I believe, may even so stimulate the peristaltic action of the gall-bladder, that it may suddenly contract with sufficient force to cause the immediate extrusion of a gall-stone from it, and at once give rise to an attack of acute biliary colic,

from the stone being suddenly thrust into the cystic duct. To be followed in a few days afterwards by a true jaundice, from the stone descending into and obstructing the common bile-duct.

Moreover, it has been repeatedly hinted at by writers on hepatic disease that primary cancer of the liver, in the predisposed, may have been the direct result of prolonged mental worry or protracted grief.

One of the reasons, no doubt, why jaundice does not more frequently follow upon unusual mental emotion, is simply that a certain amount of pigment is required in order to produce a visible tinging of the body, and it seldom happens that the emotional effect on the biliary secretion is sufficiently great or permanent to permit of the requisite amount of pigment accumulating in the blood. The reason, too, why mental emotion is more apt to cause jaundice immediately after a meal is, as will afterwards be better understood, that the congested state of the liver at that time favours the stoppage of the secretion. A blow on the head, which is now and then observed to be suddenly followed by jaundice, acts, I believe, in the same way as fright, namely, by paralysing the nerve force required for the continuance of the biliary secretion. Just as happened in the case of the dog with the biliary fistula, which I struck on the head, related at page 76.

CHAPTER IX.

JAUNDICE FROM ACTIVE HEPATIC CONGESTION.

As this is one of the commonest causes of jaundice from suppression, and many different varieties of active hepatic congestion have been described by home and foreign medical authors, in order to make myself easily understood I shall require to say a few words on each of the different forms separately, paying greater or less attention to them in exact proportion to their clinical importance.

In all cases where the congestion of the liver exists to a sufficient extent to produce jaundice, the organ is found to be appreciably enlarged ; and the first variety of the congestion I shall consider is that named

Hepatitis.

The most striking sign of hepatitis is jaundice ; and the mechanism of jaundice arising from either the acute or chronic forms of hepatitis, as well as from all the other varieties of active congestion

of the liver, is readily explained on physiological grounds. Which are :—

A congested condition of any gland, whether amounting to actual inflammation or not, is unfavourable to secretion. We know, for example. that congestion of the kidney is accompanied by a suppression of the urinary secretion, and that the secretion is gradually re-established, *pari passu*, as the congested condition of the organ diminishes. The suppression of the renal secretion is no doubt due to the engorged capillaries pressing upon the secreting structure and ramifications of the urine tubes, and thereby annulling their functions. A similar explanation is equally applicable to the biliary secretion ; and just as it happens in the case of the kidney, that it is exceedingly rare for a total suppression of its functions to take place, so with the liver it seldom happens that the congestion is sufficiently universal to induce complete arrest of the biliary secretion. We find, therefore, that although there may be sufficient to induce a yellowness of the skin and high-coloured urine, pipeclay stools are frequently absent in such cases. Sufficient bile to tinge the fæces still being secreted by a portion of the liver, and finding its way naturally into the intestines, prevents the stools from becoming pipeclay-coloured.

Undoubtedly it must have occurred to many of

my readers, that jaundice is even frequently absent in severe cases of acute inflammation of the liver—even those running on to suppuration—and that, consequently, the foregoing theory of the pathology of such cases is insufficient. At one time I was puzzled to explain this apparent anomaly, but on subsequent investigation the true cause became apparent, and instead of the above facts detracting from, they only tended to strengthen the theory. For when we closely examine even severe cases of acute hepatitis without jaundice, we invariably find they are those in which only a portion of the liver is affected. It matters not whether it be one lobe or two, the surface or the centre of the organ: the disease is invariably found to be circumscribed, and enough hepatic tissue left in a sufficiently normal condition to prevent the constituents of the bile accumulating in the blood and producing jaundice. This may, and often does, occur, even when the disease has run on to suppuration. The abscess, or abscesses, being limited entirely to one portion of the liver.

The most typical example of jaundice as the result of active congestion is to be found in those cases where it supervenes on an attack of subacute hepatitis, such as is met with in hot climates, where indolent habits and high living favour portal congestion. It is occasionally also met with in England,

however, and is then generally associated with considerable gastric derangement.

I had occasion to witness a good example of this form of subacute active hepatic congestion in the person of a French gentleman, who was brought to me some days after his arrival in England, on account of his skin having assumed a most intense yellow hue. It appeared that he had come to England on a visit to friends, and, rather enjoying the novelty of an English table, indulged too freely in a quantity and quality of food and drink to which he had hitherto been a stranger. The consequence was, that within three or four days after his arrival, he began to suffer from dyspeptic symptoms and hepatic tenderness. His skin at the same time assumed a dusky hue, which soon merged into a decided yellowness. These symptoms were accompanied by saffron-coloured urine and pipeclay stools. On the urine being tested it gave a distinct bile-pigment, but no bile-acid reaction—a point which, I shall afterwards have occasion to show, is of great diagnostic value in obscure cases of jaundice. This gentleman, under the influence of low diet, blue pill, and benzoic acid, perfectly recovered his normal complexion in the short space of a week.

The symptoms of all the varieties of hepatic congestion being in their main features the same, and only differing slightly from one another in individual

cases, I shall give them here, once for all, in a well-marked form, and then I shall only require to call attention to the characteristic peculiarities they exhibit in the special variety happening at the time to be under consideration.

General Symptoms of Hepatitis.

1. A sense of fulness and discomfort in the hepatic region, in some cases amounting to actual pain. 2. Tenderness on percussion, and acute pain on firm pressure. 3. More or less increase of dulness in the perpendicular right nipple line. Instead of the dull extent being the normal four, it may be as much as six, eight, or even, in rare cases, ten inches. 4. Yellow, hot, and dry skin, with pyrexia. 5. Yellow conjunctivæ, foul tongue, and rapid pulse. 6. Urine scanty, saffron-coloured, and, on cooling, turbid, with a great deposit of lithates—ochre, pink, or red coloured —sometimes albuminous, but always (except when kidney disease also exists) of a specific gravity of over 1015. 7. Light- or pipeclay-coloured stools.

If these symptoms are present, there need be no hesitation felt in diagnosing the case as one of jaundice from suppression in consequence of active congestion of the liver.

General Treatment.

The first thing in a case of acute hepatic congestion is to enjoin strict rest. The second is to put the patient on low diet. The third to freely clear out the bowels. The fourth to relieve the slight discomfort by the application of hot, thick, and large linseed poultices. The fifth, if there be signs of acute inflammation, to apply a freezing mixture of ice and salt or leeches or even cupping-glasses over the painful hepatic region. It is sometimes surprising how speedy and complete is the relief afforded by free local depletion. As may be judged of from my remarks on general therapeutics, podophyllin and the whole of the other forms of hepatic purgative stimulants, without one solitary exception, are totally inadmissible in all cases of active hepatic congestion. Mercury is here, as in many other instances of hepatic disease, our sheet-anchor, both in the shape of a purgative and an antiphlogistic, for the reasons already fully given in the chapter specially devoted to its consideration, and to which, in order to save repetition, I beg to refer the reader (page 157).

Peri-hepatitis.

Peri-hepatitis is the name applied to those cases where not the parenchyma, but the capsule surrounding the parenchyma alone, is inflamed. That the capsule of the liver is often inflamed by itself, the

parenchyma remaining unaffected, I much doubt. But that both are at one and the same time inflamed is, I believe, a matter of frequent occurrence, and one readily knows when this is the case by the friction sound distinctly audible when the stethoscope is applied in the right hypochondriac region. The friction sound is produced during the act of inspiration by the rubbing of the inflamed serous covering of the upper convex surface of the liver on the sympathetically affected lining serous membrane of the under concave surface of the diaphragm. In these cases, the application of the stethoscope in this region reveals a friction sound just as readily and quite as audibly as in acute pleuritis.

If an hepatic friction sound exists without jaundice, or even without so much as a sallowness of the skin, then the case may be safely diagnosed as one of peri-hepatitis ; but if the friction sound is associated with a yellow discoloration of the skin, it shows that the parenchyma as well as the capsule of the liver is affected, and that the case is consequently not one of peri-hepatitis pure and simple, but one of general hepatitis, with the inflammation extending to the capsule of the organ.

Sympathetic Hepatitis.

Sympathetic inflammation of the liver associated with distinct jaundice has been known to supervene

upon an attack of pneumonia of the base of the right lung. The pathology of this condition is simple, for there cannot be any doubt that it is due not to the transposition of morbid matters, but to a sympathetic inflammatory action being excited in the liver, or perhaps it would be more correct to say *extended to the liver* through the diaphragm from the inflamed lung by the pneumogastric and sympathetic systems of nerves. The extension of this inflammatory action, or even its supervention in the liver by sympathy from the inflamed lung, is rendered all the more comprehensible from the known anatomical fact that both the liver and the right lung are freely supplied by branches of the same pneumogastric nerve, and, as we well know from our experience with eyes, that direct nerve communication favours sympathetic inflammatory action.

I may mention that in the 'British Medical Journal' of 1868, Dr. Cheadle relates a strange case of a girl aged six, who, while she was labouring under jaundice, was seized with pneumonia of the upper left lobe. When immediately the jaundice disappeared, and as speedily returned after the inflammation of the lung subsided, and remained for some weeks afterwards.

The connection between general pulmonary disease and jaundice has not yet been properly accounted for; nor will it be so until the subject has been more

fully investigated than it has hitherto been. We have too many theories and too few facts in hand at present to admit of my generalising on the subject. But in connection with this matter I would call attention to a fact very little known, though it was pointed out by Alexander Shaw in the numbers of the 'Medical Times' of 15th July and 30th September 1842, which is that the portal circulation is greatly influenced by the respiratory movements. The mere expansion and contraction of the thoracic walls being sufficient of itself to propel the blood through the portal vessels, by as it were a suction force. This being the case, it is very easy to understand how in cases of pneumonia, when the movements of the chest are greatly impeded, a stagnation of the portal blood may occur in the liver and induce sufficient hepatic engorgement to produce jaundice. This observation of Shaw's explains, too, the beneficial effects of violent exercise, which increases the respiratory functions, in cases of torpid liver.

Not only does thoracic but abdominal inflammation sympathetically affect the liver. Thus M. Hervieux has shown that an attack of jaundice may supervene in cases of peritonitis following upon delivery. He published, in 1867, a *brochure* entitled 'Ictère puerpéral,' which he commences by relating the case of a woman, aged thirty-four, who six days after delivery (while suffering from acute peritonitis)

was seized with an attack of well-marked jaundice, and died two days afterwards. At the post-mortem the liver was found stained yellow, soft, fatty, and the cells granularly degenerated, as in acute atrophy, but not to such a marked extent, nor was the volume of the liver diminished. Even the kidneys, as well as the liver, were stained yellow by the bile-pigment. This case I specially refer to, as it is the most typical one he cites of jaundice sympathetically induced by abdominal inflammation, and from its being the one upon which his observations chiefly hinge.

Interstitial Hepatitis.

This supposed *special* form of inflammation of the liver is said to merit the name of interstitial in consequence of 'an inflammatory hypertrophic condition of the interlobular cellular tissue being the principal structural change observable in the tissues of the liver.' But as the interstitial tissue—that is to say, the connective tissue—of every inflamed glandular organ presents a precisely identical state of things, and ordinary hepatitis itself offers no exception to the rule, the mere finding an excess of white blood-corpuscles in the inflamed connective tissue (which said excess of white blood-cells, we are told, is the sole diagnostic characteristic of interstitial hepatitis) appears to me scarcely a sufficient pathological reason for dubbing even this, *supposed to be*,

special inflammatory condition interstitial hepatitis: especially seeing not only that the difference in its histological conditions from those of ordinary hepatitis is at most merely a difference of degree, but that the functional biliary derangements it produces are perfectly identical with those produced by ordinary hepatitis—namely, jaundice from suppression. Dr. Borelli,[1] however, actually goes so far as to say that he can distinguish the one form of hepatitis from the other, even during the lifetime of the patient, by the peculiar circumstance that in interstitial hepatitis the dulness increases upwards, towards the nipple, instead of—as in ordinary hepatitis—downwards, into the abdomen. A peculiarity which he attributes to the non-resistance of the diaphragm, from the interstitial inflammation of the liver extending itself to it and weakening its powers of resistance. I give this idea of Dr. Borelli's without comment, for the simple reason that I fail to grasp it, and consequently will leave my readers to accept or reject it as they think fit.

These are the four forms of hepatitis which have been specially named from their supposed pathological characters. The varieties next to be considered are those that have derived their titles from the most prominent factors in their symptomatology.

In one respect all the members of this latter

[1] *Würzburg. Arch. Med.-Phys. Gesellschaft*, Bd. viii.

group closely resemble each other. Namely, in that they are all more or less directly traceable to the introduction into the body of a toxic agent, animal, vegetable, or mineral—to wit, of true fermentative living animal micrococci and bacteria; of living vegetable miasmatic and malarial germs; the poison of snake-bite, of fish, and of fungi, as well as of dead mineral poisons, such as phosphorus, lead, and antimony.

Before proceeding to consider each of these exciters of active congestion of the liver in detail, I beg to call the reader's special attention to the fact that good reasons will presently be given for believing that all the various and, at first sight, apparently different varieties of liver affection, arising from the direct introduction into the human system of toxic agents—so widely differing from each other as pyogenic, epizootic, miasmatic, and mineral—have an identical structural pathology, and that, too, even when they assume the apparently distinct forms of either endemic and sporadic, or epidemic and contagious diseases. It seems as if Nature acted here, as she does everywhere else, on one great and uniform plan. This is rendered all the more evident when we compare together cases so apparently widely differing from each other as the sporadic acute atrophy of the liver of temperate zones with the contagious epidemic jaundice (yellow fever) of the

tropics, which, at first sight, appear as different as midnight from noonday, and yet, on nearer inspection, are found to resemble each other so closely as to force upon us the conviction that they are in reality mere varieties of precisely the same morbid state, only differing from each other as regards degree of contagiousness; which probably, again, is solely regulated by the temperature of the locality in which the disease occurs. Acute atrophy, though generally sporadic, sometimes assumes the form of a miniature contagious epidemic. Contagious jaundice (yellow fever), on the other hand, although it generally sweeps in great epidemic waves over the districts it infests, sometimes appears in the form of mere isolated and but slightly contagious cases.

CHAPTER X.

JAUNDICE, THE RESULT OF HEPATIC DISEASE, CAUSED BY DISEASE-GERMS.

Although it has long been known that certain forms of large parasites produce hepatic disease—to wit, hydatids, flukes, and round worms—it will no doubt surprise some of my readers to learn that the minute parasites which are denominated germs are sometimes also the producers of serious and even fatal forms of hepatic complaints. Dr. Evans, as I elsewhere (p. 449) show, found that an epidemic of jaundice among horses was the direct result of the presence of minute filaria in their blood-vessels ; and now I am about to add another interesting fact to the etiology of hepatic derangements by pointing out that minute microscopic organisms, both of an animal and vegetable type, are the cause of various forms of pyrexial hepatic diseases.

There are several apparently widely different forms of hepatic disease which I believe to be directly traceable to the introduction into the circulation of epizootic (animal) and miasmatic (vegetable) germs ; and being now at the chapters specially devoted to

the contagious and epidemic forms of liver derangements, I shall take occasion, as I proceed, to call special attention to a point in their etiology and pathology hitherto unnoticed. Namely, the important part germs not only play in the production of many hepatic diseases, but in the production of the pyrexia and brain symptoms accompanying the multitudinous forms of tropical as well as temperate hepatic derangements. Not only associated with specific, typhus, scarlet, and malarial fevers, but with acute atrophy, septicæmia, pyæmic and metastatic, abscesses, &c. I believe that tropical contagious jaundice—the so-called yellow fever—the temperate zone acute atrophy of the liver, pyæmic hepatitis, &c., are one and all of them due to epizootic disease-germs of the bacilli type. A full account of whose nature I gave in the 'Lancet' of June and July 1881.

While all the various forms of malarial hepatitis and jaundice are equally, I believe, due to the presence in the body of disease-germs of a vegetable type. Either in the form of micrococci or bacteria. When the etiology of many hepatic diseases is so regarded, the difficulties hitherto encountered in finding a logical explanation, not only for the occurrence of isolated sporadic cases, but of the waves of hepatic epidemics which ever and anon sweep over the surface of the globe in different forms at various times, ceases to be an inscrutable pathological problem.

While the albuminoid fermentative germ theory moreover, with its known development of multitudinous animal and vegetable microscopic organisms, not only satisfactorily explains the cause of an incubation

FIG. 5.

Disease-germ spawn.

FIG. 6.

Bacilli disease-germs.

period, but likewise in an equally satisfactory manner the intermittent character of all epidemics of hepatic disease. As I fully explained all this in the 'Medical Times and Gazette' of November and December, 1881, I need not do more here than say that in the

FIG. 7.

Micrococci.

FIG. 8.

Bacteria.

first place the germs admitted into the circulation always require a certain time to develop and multiply before they can produce sufficient toxic effects to admit of our applying to them the name of disease, and that we all know, although the period of incuba-

tion is, as a general rule, well defined in each particular form of disease, it is nevertheless liable to great variations. Ay, I believe to far greater variations than is in general supposed. For example, it will probably startle some of my readers who do not accept the theory of contagious hepatic disease-germs, and even some of those who do, who are not, however, well versed in the life-history and mode of action of disease-germs, to learn that those producing malarial hepatitis often lie dormant in the patient's body for one or two years before producing any visible signs. As my papers in the 'Medical Times and Gazette' give a full explanation of this, at first sight, strange anomaly in the history of disease, in order to save space by not repeating the data I have collected, I beg to refer my readers to the series of papers above alluded to, both in the 'Lancet' and 'Medical Times' of 1881, and at once proceed to the consideration of

The Epidemic Jaundice of Temperate Zones.

It is seldom that jaundice attacks persons in an epidemic form in temperate zones, but it does so occasionally, and that too in almost all parts of the world, Europe, Asia, and America. For the last hundred years and more, epidemics of jaundice have been at various times recorded, and recently in the 'Recueil de Mémoires de Médecine Militaire,' vol. iii.

p. 374, M. Martin gave an account of an epidemic of jaundice which he had the opportunity of observing among the artillerymen and engineers of the French army stationed at Pavia during the last Italian war. The epidemic commenced during the great heats of August, and terminated in the end of October. There occurred 71 cases in an effective of 1,022 men. The causes which he considered gave rise to the congestion of the liver were the unusual heat, the fatigue of long marches, the indulgence in alcoholic drinks, and the marsh miasmata prevalent in the district.

Great increase in the size of the liver was observed in most of the cases, and of the spleen in many ; all complained of pain in the epigastrium and in the hypochondria. In fact, the onset of this last was the first symptom of the approaching jaundice. None of the cases proved fatal. Professor San-Galli informed M. Martin that a similar kind of epidemic prevailed among the inhabitants of the town of Pavia itself at precisely the same time. M. Martin's theory of the cause of the epidemic among the soldiers requires revision before being implicitly accepted. For if a similar epidemic occurred in the town, the inhabitants of which were not subjected to the same influences, coupled with facts which I shall presently adduce to show that heat is by no means an essential factor in the production of epidemic jaundice, M. Martin's theory falls to the ground.

In order to prove that heat is by no means an essential to epidemic jaundice, I may refer :

First, to the history of an epidemic of jaundice of six months' duration, which occurred near Offenbach in the winter of 1874–5, recorded by Dr. Klingelhoeffer in the 'Berliner Klinische Wochenschrift' of February, 1876, in which he states that both sexes were equally affected, though none under twenty years of age, and that the jaundice appeared to him to be due to catarrh of the bile-ducts.

Secondly, to what is further stated in the 'Lancet' of February 21, 1863, under the head of the 'Health of Rotherham,' that 'in last November scarcely had a fatal epidemic of fever subsided ere another, less fatal, but as widely spread, took its place. Several persons were attacked with jaundice, and now not less than 150 persons are suffering from it. None of those who were attacked by the late fever are suffering from the present epidemic.'

Thirdly, in the cold months of January and February, 1869, an epidemic of jaundice occurred in Dublin, and was brought under the notice of the Dublin Medical Society by Dr. Haydon in a paper which he entitled 'An Epidemic of Jaundice,' &c.

We thus see that heat, though it may be, and most probably is, a favouring cause, is by no means an essential factor in the production of epidemic

jaundice. As bearing also on the etiology of the disease, I may,

Fourthly, further state that M. Corville has given an account of an epidemic of jaundice among the inmates of a prison, which occurred during the summer of 1859, in which eleven persons died out of a total of forty-seven that were attacked; which gives the high rate of mortality of 23 per cent., and makes it, in this respect, approach the epidemic jaundice of the tropics. ('Arch. de Méd.,' 1864.)

Fifthly. Before leaving the subject of epidemic jaundice in temperate zones, I have again, as I did twenty years ago, to call special attention to the fact that jaundice may, and does occasionally, occur in an epidemic form among pregnant women. But as attacks of jaundice sometimes come on in pregnant women without assuming an epidemic form, I shall briefly allude to them first.

Jaundice of Pregnancy.

It has been long noticed that sporadic cases of jaundice are now and again met with where the pregnant condition of the uterus appears to be the sole exciting cause of the attack. For example, Dr. Gooch in his work on diseases of women (Sydenham Society's edition, p. 56) relates the case of a lady who became jaundiced after three consecutive confinements. Her case is peculiar, for her first

attack of jaundice might be attributed to sudden nerve-shock, caused by a house taking fire close to hers. The shock was sufficient to induce a temporary attack of mania, and might therefore be supposed to have been sufficiently severe to produce jaundice in the manner described at page 310. But fifteen months afterwards she was again confined, and eight days later temporary jaundice showed itself, but unassociated with cerebral symptoms. While she again became jaundiced *before* her third confinement. It was completely cured before delivery by the administration of purgatives.

This may be considered as a good example of jaundice occurring as the result of pregnancy.

It thus seems as if the mere normal function of gestation in the human female predisposes to attacks of jaundice much in the same way as it predisposes to attacks of Asiatic cholera. Indeed, in more ways than one, the peculiar condition of system to which the pregnant state gives rise in the human female seems to render the body specially liable to hepatic derangements of a very grave character. For example, it is now a well-recognised fact that the fatal disease known under the name of acute atrophy of the liver not only more frequently attacks women in the early months of pregnancy—at which time their nervous systems are more prone to both mental and physical impressions than at any other

—but is also more frequently followed by fatal consequences than when it attacks non-pregnant individuals. I need not at present dilate further on this point, as I shall again have occasion to refer to it in the chapter specially devoted to the consideration of acute atrophy. Meanwhile, I need only further remark that it is not only acute atrophy, but also other conditions of liver, giving rise to ordinary jaundice, which are common in the early months of pregnancy. So that it may be unhesitatingly said that there is an undeniable intimate connection between affections of the liver and the condition of the system resulting from pregnancy; the only question being, how is this connection to be explained? Is it due to direct nerve influence—like sympathetic hepatitis—or does it depend on some other cause?

In a letter from Mr. J. J. Frederick Barnes, which appeared in the 'British Medical Journal' of January 24, 1880, it is suggested that the jaundice of pregnancy is due to 'the vital force of the economy as a whole not having sufficient potentiality for the due performance of the animal functions under the access of fresh conditions caused by the pregnant state. The liver, owing to its proximity to the disturbing factors, suffering local congestions and other abnormal interferences with its functions, is the organ most likely to yield to the pressure put upon it. We thus

have a previously bad state of health rendered worse by the disturbance of function of the hepatic machinery and the consequent imperfect performance of the digestive process, ending in impaired nutrition and a condition of anæmio-chlorosis.'

Some others again have gone even so far as to suggest that normal pregnancy is associated with abnormal parenchymatous glandular degeneration. Such an idea, however, is contrary to all the laws of nature. To suppose that a normal gives rise to an abnormal process in a healthy frame, would be simply a solecism in the interpretation of organic structural as well as functional law. An idea only tenable by a mind totally unversed in scientific medical philosophy. The etiology of the connection between pregnancy and liver disease appears to me much more likely to be found in the peculiar condition of blood and nervous system to which the fœtal development and the rapid evolutionary changes in the uterus give rise during pregnancy. More especially in the earlier months, when, as is well known, there exists extreme irritability of the nervous system, associated with a supersensibility to mental and physical impressions. At the same time, I must here call attention to the important fact, that not only may a jaundiced woman become pregnant, but a pregnant woman become jaundiced quite independent of the existence of any apparent direct connection between the hepatic

organ and the fœtal development and uterine evolutionary changes taking place during the pregnant state. Just as a man may happen to have a broken leg and a black eye at one and the same time, without there being any direct connecting link between their exciting causes. It is, therefore, necessary for the young practitioner to guard carefully against confounding together a case of jaundice occurring *during* pregnancy with one of jaundice occurring as the *result* of pregnancy. The being able to make a differential diagnosis of these two kinds of cases is highly important; for while in the one case it is the jaundice alone to which particular attention requires to be paid, in the other it is the condition of the uterus which demands special care; for abortion, miscarriage, or premature labour is the usual sequel of the latter class of cases. This is, unfortunately, about all that can be said on the matter, for we have no proof whatever that the pregnant uterus *per se* acts prejudicially on the liver's functions either mechanically or physiologically. For the enlarged uterus cannot possibly be said to exert a deleterious pressure on the liver, either as a whole or partially. Otherwise every, or nearly every, pregnant woman's liver would get out of order in the last few weeks before delivery. Besides which, every enormous fibrous tumour of the uterus, as well as every large ovarian cyst, would be associated with hepatic derangements. Whereas, so

far from this being the rule, it is in reality the exception.

That there are still believers in the theory that the pressure of the pregnant uterus may induce jaundice, is not to be wondered at, when men like Dr. Litten believe in the pressure of a movable kidney being capable of doing so. He reports in the 'Charité-Annalen,' Band v., 1880, the history of the case of a woman thirty-seven years old, in whom he believes that repeated attacks of intense icterus were produced by the pressure on the gall-duct of a movable kidney.

I shall now proceed to give an example of

The Epidemic Jaundice of Pregnancy,

before I attempt to explain its pathology, or sum up my views on the *rationale* of the epidemic jaundice of temperate zones.

That jaundice may occur in an epidemic form among pregnant women was conclusively shown by Dr. Saint-Vel, who relates that 'in 1858 the island of Martinique was, without appreciable cause, visited by an epidemic of jaundice, remarkable for its severity in pregnant women. It broke out at St. Pierre towards the middle of April, attained its maximum height in June and July, and terminated towards the end of the year. All races were attacked; the patients were mostly adults; no liver complication

could be detected; nor could any resemblance be traced between the disease and yellow fever. It was fatal to females only, especially during pregnancy. Of thirty pregnant women who were attacked at St. Pierre, ten only arrived at the full period of pregnancy without presenting any other symptoms than those of ordinary jaundice. The other twenty all had abortion or premature labour a fortnight or three weeks after the commencement of the attack, and died in a state of coma, which appeared a few hours before or after the expulsion of the fœtus. The females who died were from the fourth to the eighth month advanced in pregnancy. In some cases, slight delirium preceded the coma, which was never interrupted, but became more and more profound up to the time of death. Its longest duration, which was only in two cases, was twenty-four and thirty-six hours. It was not preceded by any notable modification of the general sensibility, or of the respiration or circulation. Hæmorrhage was absent, except in one case, where a female had it before delivery. When death was delayed till three or four days after delivery, the lochia were healthy. Almost all the children were still-born; some lived a few hours; one alone survived. None of the infants had the icteric colour; nor was there any other sign of jaundice whatever in the ten children born at the full term.'

The foregoing translation, which is from the

'Gazette des Hôpitaux,' November 20, 1862, appeared in the 'British Medical Journal' of February 7, 1863, p. 141.

Notwithstanding that the epidemics of jaundice which occur in temperate climates are not limited to the hot months of the year alone, nor usually present contagious symptoms, nor are as a rule very fatal, yet, as they are occasionally associated with hæmorrhages from both stomach and bowels, are occasionally contagious, and have sometimes a high mortality (= 23 per cent.), I have no hesitation in placing them in the same category as the contagious epidemic jaundice of the tropics, which I am now about to describe, and of which I regard them as being only a less virulent form, in consequence most probably of milder climatic causes. The value of this theory will become more apparent after the chapters on contagious jaundice and acute atrophy of the liver have been perused.

The Epidemic and highly Contagious Jaundice of the Tropics.

There are two perfectly distinct forms of tropical jaundice, which have been described in books as yellow fevers. They are in general spoken of as :—

A. Febris Icterodes = Specific Yellow Fever.

B. Febris Icterodes Remittens = Malarial Yellow Fever.

The difference between these two forms of disease

is, that while the first is a form of epidemic jaundice accompanied by continued fever, the result of epizootic disease-germs, and propagable by contagion both in temperate and tropical regions, though only producible *de novo* in the latter, and only occurs once during the lifetime of a patient, the second is a form of non-contagious epidemic jaundice associated with fever of a distinctly remitting type, the result of miasmatic disease-germs, transportable by a patient into temperate zones, but not producible there, and liable to recur several times in the same individual. These two forms of epidemic jaundice possess the following points of similarity :—

1. They both originate in tropical countries only.

2. They are both capable of being transported by infected individuals into temperate zones.

3. The liver is the glandular organ mainly at fault.

4. The kidneys are sympathetically more or less affected.

5. Black vomit and tarry stools are their usual accompaniments.

6. Their febrile symptoms are moreover said to be so very much alike that when a sporadic case of either occurs within the geographical range in which both forms of the disease are known to be indigenous, it is said to be not only difficult, but frequently impossible, to distinguish which form of the affection

the patient is labouring under. A fact which appears all the more extraordinary when it is known that these two forms of jaundice, though possessing such well-marked common febrile symptoms, have diametrically opposite exciting causes, namely :—

The first, or Febris Icterodes, is essentially due to the introduction into the system of a highly contagious epizootic germ poison.

The second, or Febris Icterodes Remittens, is, on the other hand, as palpably due to the introduction into the system of a non-contagious malarial germ poison.

Lastly, I may observe that these two pathologically distinct diseases were christened yellow fevers at a time long before their pathology was known, and derived the name solely from their possessing the common signs of yellow skin and febrile disturbance.

With these preliminary remarks I shall now proceed to show, by the aid of modern research, that the so-called yellow fevers of the tropics are nothing more or less than ordinary cases of jaundice from hepatic disease of a more than usually severe type, and that we possess in England their exact counterparts, in at least a sporadic, if not even in a merely milder epidemic form, on account of a high temperature being one of the essentials in their production. I expect even to be able to show this so clearly that after I have fully explained the whole facts of the

case, I believe that if the symptoms of either form of the affection were presented to the reader, disassociated from the words tropical and yellow fever, and he were required to give an opinion regarding the nature of the case, he would spontaneously and unhesitatingly put it down as one of jaundice from liver disease, without the idea of its being one of tropical yellow fever having ever so much as crossed his brain. So I have very little doubt that after he carefully peruses all the facts I am about to lay before him regarding the pathology as well as the symptomatology of the so-called tropical yellow fevers, he will coincide with me in saying that the name of yellow fever ought to be expunged from our nosology, as being both clinically and pathologically an incorrect definition of the morbid condition the name is intended to convey, and that the word JAUNDICE should be substituted. For, as will immediately be shown, the titles which both clinically and pathologically correctly define these diseases are :—

A. Contagious Jaundice = Febris Icterodes.

B. Malarial Jaundice = Febris Icterodes Remittens.

The following are my reasons for abandoning the misleading titles of specific and malarial yellow fevers.

a. The so-called specific yellow fever not only resembles, in so far as yellowness of the skin and conjunctivæ is concerned, one and all of the ordinary forms of jaundice from suppression, but is likewise

analogous to them in the liver being enlarged and tender.

b. The pyrexia, delirium, and suppression of the urine, which are spoken of as being characteristic signs of specific yellow fever, are not uncommonly met with in bad cases of jaundice occurring in temperate zones.

c. Both in some severe cases of acute atrophy, snake-bite, and emotional jaundice, the signs and symptoms are occasionally as sudden in their onset, as brief in their course, and as rapidly fatal in their termination as even the most virulent cases of yellow fever.

d. In acute atrophy, in virulent snake-bite, and in emotional jaundice of temperate climates, hæmorrhage into the stomach and bowels is a common symptom, producing the appearance of the black vomit and the tarry dejections of tropical yellow fever.

e. In acute atrophy, exactly as in yellow fever, the skin so completely assumes the functions of the kidneys as to cause the sweat to acquire a distinctly urinous odour.

f. The only point of dissimilarity observable in severe cases of acute atrophy and the so-called specific yellow fever is that while the one occurs sporadically in temperate climates, the other occurs epidemically in the tropics.

g. Specific yellow fever, or, as I prefer to call it,

contagious jaundice, resembles very closely the jaundice associated with the typhus and scarlet fevers of temperate zones, in so far as they are all distinctly due to the introduction of specific forms of septicæmic germ poison. The germs of which, when once introduced, grow, spread, and propagate themselves in the human body.

h. So-called specific yellow fever closely resembles in all its essential symptoms the severe febrile forms of jaundice, even of the remittent and intermittent types, which occur in certain parts of Africa and Asia, where true yellow fever is supposed to be totally unknown. The only difference between the two sets of cases being that the one is highly contagious and the other is not.

i. In order still further to substantiate my view that specific yellow fever is nothing more or less than a mere form of *true* jaundice, arising from a disordered condition of the liver consequent upon the accidental introduction into the circulation of an epizootic germ, I shall here cite a case of supposed yellow fever which was reported in the 'Lancet' of July 22, 1865, by Surgeon Gabriel of the Royal Navy, in which the disease was associated not only with hepatitis, but with psoas abscess.

The patient, a sailor, aged 23, came under his treatment at Nassau, N. P., on December 7, 1863, suffering with the symptoms of acute hepatitis.

'Under the usual treatment for that affection the symptoms were successfully combated. On the third day the patient no longer experienced pain, became cheerful, and could lie in any posture without inconvenience. On the fourth day the belly was observed to be somewhat distended, and the patient complained of the presence of flatus. No pain was experienced over the abdomen under ordinary palpation, and only a sense of uneasiness under firm pressure. Pulse 100, weak. In the morning (the sixth day of illness) a severe pain occurred in the abdomen, and the patient looked much distressed, and groaned. Hot water was procured and the belly well fomented, while a dose of brandy and morphia was administered warm. The patient's countenance suddenly assumed a collapsed expression; at the same moment a coffee-ground-like vomit escaped from his mouth, and he expired.

'On examining the body seven hours after death, there was a lemon-yellow tinge over the surface generally and the conjunctivæ. (The clearness of the skin and conjunctivæ from yellowness previous to death had been particularly noted.) On opening the abdomen, the peritoneum was seen to be marked here and there with patches brightly injected, indicative of recent morbid action. Within the peritoneum purulent fluid was found in profuse quantity.

'On reaching the right iliac region the source of

the pus was at once revealed: the great psoas muscle was represented by a band of white fibrous matter so soft and yielding that the fingers could be gently passed through its substance. The iliacus was in a similarly degenerated condition; and in the course of the vessels a cavity extended into the thigh for six inches. The lumbar and a few dorsal vertebræ were *cursorily* examined (thermometer 87°), but no disease was detected in them.

'On examining the stomach, it was found filled with black vomit. Yellow fever formed an element in this disease, and a cause is not wanting to account for it. A merchant steamer was lying ahead, in which sixteen cases of fever had occurred, of which nine had died with black vomit.

'Previous to his fatal attack, the patient had been a month under treatment for a hypertrophied condition of the tissues of the thigh and iliac region; the flexure of the groin was obliterated, and the part between the thigh upwards to the abdomen was almost of uniform level. The patient complained only of the difficulty he experienced in the progression of the extremity.'

j. Again, as regards the pathological conditions of the hepatic organ in the so-called yellow fever, I may mention that Drs. Leggatt and Greenfield have, in the eleventh volume of the Clinical Society's 'Transactions,' reported a fatal case of the disease in a military

man, aged 52, who died in England (in whom the period of incubation appeared to have been twenty-five days). In which the liver was carefully examined microscopically, and is said to have presented the following appearances.

The outline of the lobules was normal. Under a low power, they were found to be in many places separated by exudation. Each lobule showed at parts bright yellow staining. In some at the centre. In others at the periphery. Under a high power, the exudation in the portal spaces was found to consist of leucocytes. Some of the bile-ducts were filled with swollen epithelium. Some of the liver cells retained their normal shape and arrangement. But they were very granular and pigmented. In many places, only a confused mass of cell-fragments mingled with nuclei were to be seen. The number of the nuclei appeared to be increased. The changes seemed to be the result of simple inflammatory interstitial exudation (which was most marked at the periphery of the lobules), and consisted of swelling of the cells, multiplication of the nuclei, fatty and pigmentary degeneration with disintegration of the cell-walls—'in fact a true parenchymatous and interstitial hepatitis.' This description reads exactly like one of the histological condition of the liver in cases of acute atrophy. (See pages 404–409).

k. I have yet to call attention to another condition

of the liver which tends to throw light upon the true nature of the so-called yellow fever. It has received the somewhat extraordinary title of Emphysema hepatis, and is described by Dr. Meigs of Pennsylvania as a complication of the indigenous enteric fever of his district. The case is published in the 'Philadelphia Medical Times.' The patient was a sailor, twenty-five years of age, who exhibited well-marked symptoms of enteric fever, which terminated fatally after profuse hæmorrhage from the bowels. At the autopsy, made eleven hours and a half after death, the upper part of the body was found swollen from subcutaneous emphysema—a condition that had not been observed before death. The liver was enlarged, and its tissue everywhere cribriform, crepitant, and spongy. Pieces cut off from it floated in water. The weather was not very hot, and no general decomposition of the tissues was present. Louis, in his work on Typhoid Fever, states that he has never seen this condition in fever, but remarks that he has met with it three times in patients dying of other acute diseases. While again, Frerichs alludes to emphysema hepatis as a local process of disintegration originating in some of those complicated metamorphoses which occur in the liver from the presence of large quantities of hydrocarbons.

Treatment of Contagious Jaundice.

Even the very forms of treatment recommended for specific yellow fever by our greatest authorities on the subject add support to the view of its being a mere contagious form of jaundice, and, like all other forms of jaundice, originating in a derangement of the biliary function depending upon physical changes in the secreting cells of the liver.

For example, Dr. Blair, the well-known writer on this subject, speaks of 'aborting the attack'—that is to say, in more poetic language, 'nipping the disease in the bud'—by giving a scruple of calomel and a scruple of quinine for a dose, and following this up with a drastic purgative, consisting of two ounces of the sulphate, along with two drachms of the carbonate of magnesia, in peppermint water. This indeed looks like heroic treatment, and I presume is more likely intended for negroes than for white men. I, for one, should certainly object to be personally subjected to it. More especially when he adds that it is sometimes expedient to repeat the dose four times in the twenty-four hours in order that the desired effect may be attained. I merely mention this form of treatment recommended for the so-called yellow fever in order to show how closely it resembles the old-fashioned system of treating jaundice in this country, where yellow fever is said to be unknown. Were I asked to give an opinion regarding the propriety of

employing Dr. Blair's heroic doses, I think I should mildly remark that I would divide each of his doses into four, and give one of them, at least as a commencement, not oftener than every six hours. Beyond that, I think I should, with my present knowledge, hesitate to go.

Dr. Blair makes another and much more pleasant suggestion, and that is, to give the patient gum water to drink (two ounces of gum-arabic in six ounces of water), in order to quench the great thirst usually complained of, until he tires of it, and then weak arrow-root or other demulcent. He further recommends enveloping the patient in a cold wet blanket. Which has the double advantage of aiding in allaying the thirst and reducing the temperature. The tenderness of the liver, which is due to hepatitis, is to be at the same time relieved by hot poultices.

When, however, there is, as frequently happens, a distinctly active inflammation of the liver, other remedies must be employed, such as leeching or cupping over the tender and painful part. Blistering in these cases must be avoided, for, as was said before, the kidneys are often sympathetically affected with a tendency to urinary suppression, and if they should happen to be so in the case under treatment, the application of a blister to the liver is almost certain to be followed by an attack of strangury which will not improbably turn out to be a grave complication in the treatment of the case.

Since, as I previously pointed out at page 207, benzoate of soda has a marked effect in removing the jaundiced tint of the skin, and Drs. Klebs, Lehnebach, and Letzerich have found it act almost as a specific in puerperal fever, I would strongly recommend a fair trial to be given to it in cases of contagious jaundice. And, as an inducement to do so, I may mention that after two out of six cases of puerperal fever (a primipara and a pluripara) had died in a few days, in spite of the energetic use of quinine and wine, in whom the temperature exceeded 109° Fahr., Dr. Lehnebach was led to try, in the remaining four cases, benzoate of soda, as recommended by Klebs and Letzerich. The result was so remarkable that he believes that, if his experience be confirmed by that of others, benzoate of soda will be as much a specific in puerperal fever as salicylic acid is in acute rheumatism.

Benzoic acid, like salicylic and carbolic acids, is a true germicide, and consequently, if my theory of animal germs being the cause of contagious jaundice be correct, it is easy to understand how benzoate of soda acts. Either benzoate of potash or of ammonia, I should imagine, would act about as well as that of soda. And when the temperature is high, the addition of the salicylate of quinine or quinine itself might be of service. The latter in about ten-grain doses.

In 1854, great excitement existed at Havannah in consequence of a Dr. Humboldt asserting that he

had found in the venom of a snake (the species not mentioned) a certain prophylactic against yellow fever. So much was it believed in that the Spanish Government gave orders that it should be practised among its troops. In consequence of which order the whole of them were inoculated with the snake venom ; and Manzini, who published in 1858 a small book on the subject entitled ' Histoire de l'Inoculation Préservative de la Fièvre Jaune,' states that, as far as his experience went, it seemed in 2,461 cases to be attended by beneficial results. While Dr. Humboldt himself asserts that during the nine previous years he had inoculated 1,438 persons, only seven of whom were attacked with yellow fever, and out of the seven only two died. Whether this be true or false, the attempt to find a prophylactic for such a scourge as yellow fever (contagious jaundice) is both highly creditable to its author, and suggestive to his reader. And if the disease be due to the presence of animal germs, it is quite possible that snake poison might act beneficially by killing the germs. Just in the same way as salicylic, benzoic, and carbolic acids do. It has even been stated that the hypodermic injection of germicides has been found to be useful. And I see no reason to doubt that, if they could be introduced in sufficient quantity into the circulation without endangering the life of the patient, the attack of the disease might be aborted.

In any case, I should strongly recommend the trial of germicides administered by the mouth. After thoroughly clearing out the stomach by the preliminary administration of an emetic, immediately followed, if it were deemed necessary, by a rapidly acting purgative, such as sulphate of soda combined with a little aromatic tincture. (Avoid the use of mercury at first. For it is too weakening in such cases, as there is always more or less tendency to hæmorrhage from the intestinal canal, either in the shape of coffee-ground vomiting or tarry dejections—the colour of both of which is due to the presence of effused and disorganised blood, and nothing else.) As soon as the stomach and bowels have been unloaded, then give a full dose of the germicide along with quinine.

Further, avoid iron in these cases, as it only aggravates the disordered state of the liver, which is in reality the organ which bears almost the whole brunt of the disease. For it seems as if the germs concentrated their attacks upon it. At first making it congested and tender. Then soft and small as in acute atrophy. Which I shall presently show is a mere sporadic and mitigated form of contagious jaundice (yellow fever).

As regards the head and general nerve symptoms in these cases—delirium and convulsions—they are in general best subdued by the free administration of the chloride of ammonium, the bromide of ammonium,

the acetate of ammonia, and the carbonate of ammonia, either given separately, or combined in pairs dissolved in camphor mixture.

Morphia has been recommended ; but owing to the supposed inaction of the kidneys, and its tendency to produce suppression both of the renal and of the biliary function, it must be employed with great caution, and even then only in very exceptional cases. Light and non-stimulating foods are to be given every two hours, along with good sound light claret in ounce doses, if the condition of the patient demands stimulation.

For further hints on treatment, consult the chapter specially set apart to its general consideration (p. 149).

I cannot quit the subject of contagious jaundice—the so-called specific yellow fever—without repeating what an intelligent layman, a patient of mine, once said to me about it. The gentleman, Mr. J. E. Naylor, bank manager of Buenos Ayres, one day, while speaking of liver disease, said to me, 'Well, do you know, I think yellow fever has more to do with the liver than anything else, for you can cure it if you only get rid of all the bile.' Then he went on to say that he was in Buenos Ayres in 1871, when 25,000 persons died of the disease. The greatest number in one day being 1,250. He said the epidemic began by a solitary case coming into the town from Paraguay,

and the disease spread and was kept up spreading by the bad drainage more than by the effects of mere contagion; for, as he remarked, it did not attack people living beyond a mile of the town, and not even a single grave-digger, who lived in the country, five miles from the town, took it; though they used to sit and smoke their pipes on coffins so badly made that the effluvium from the decomposing corpses in them was disgusting.

The first symptoms of the disease, he said, were headache, pain in the back of the neck and down the spine, and the way to stop the disease was immediately to give a sudorific and a large dose of castor oil, and keep the bowels continually going and quite empty by repeated doses of the oil, which he said brought away *enormous* quantities of *black tarry stools*, and the marvel to him was where all the 'tarry stuff' came from, as the small quantities of food the patients took would not account for it. Purge, purge, purge, was, he thought, the thing to cure. The 'tarry stuff,' as he called it, was, as I before pointed out, not bilious, but *bloody* stools. Blood during its passage through the intestines is always turned black, and is constantly being mistaken even by medical men for bilious matter; not alone in cases of contagious jaundice of the tropics, but in those of acute atrophy of temperate zones. 'Suppression of the urine,' he said, 'we regarded as a most dangerous sign. It

always began early in the disease in all the fatal cases, and until the urine again began to flow we never thought the patient safe. As soon as the urine made its appearance, however, the patients were sure to get well.'

Having, as I think, shown good grounds for expunging from our nosology the name of 'Specific Yellow Fever,' I shall now proceed to show that the title of malarial yellow fever ought for similar reasons to be abandoned; for, as will be presently seen, it is nothing beyond a severe form of ordinary

Malarial Jaundice.

Febris Icterodes Remittens is generally described in text-books as follows:—A malarial form of yellow fever originating in tropical countries, though capable of being transported into temperate zones, but incapable of being propagated by contagion. Oftentimes recurring in the same patient, and its course being marked by distinct remissions and exacerbations. Resembling specific yellow fever (contagious jaundice) in its most characteristic symptoms. Namely, a jaundiced condition of the skin associated with black vomit—hæmorrhage from the stomach. Differing from it again in the stools being rarely tarry—hæmorrhagic—or the urine suppressed, and, although headache is a constant symptom, delirium or convulsions being but rarely met with.

The malarial jaundice, no matter whether the malarial attack have been of the intermittent, remittent, or relapsing type, is invariably associated with a greater or lesser enlargement of the liver. There is usually, however, less sensation of fulness or pain complained of, and the hepatic region bears both percussion and firm pressure much better than in the majority of other cases of jaundice from active hepatic congestion. In fact, the symptoms and signs are all less marked. The skin is less yellow and hot. The febricula is less. The pulse is less quick—not usually above 90. The stools, though pale in colour, are not in general what are termed pipeclay-coloured, and the urine, though scanty and dark, is seldom of a yellowish black or deep saffron hue. Generally, though not always, however, it deposits urates on standing.

In some cases again, as I shall presently show, the urinary symptoms are among the most striking features of the malarial jaundice, and so important that I shall devote a few pages to their separate consideration (page 370).

Chronic Malarial Liver Disease.

This title includes within its capacious boundaries all the various forms of yellow discoloration of the skin arising from the different and peculiar conditions of the liver induced by one severe chronic or many slight acute attacks of malarial fever. Either with or

without distinctly intermittent aguish symptoms, in which the liver either partially or entirely for a time, at least in so far as its biliary secretion is concerned, strikes work. In the majority of such cases there is an appreciable chronic enlargement of the hepatic organ, and consequently, when there is distinct jaundice along with the other signs and symptoms, little hesitation can be felt in putting the case down as one of jaundice from suppression—in consequence of malarial hepatitis. Every now and again, however, one comes across cases where there is malarial jaundice, and yet no very distinct evidence of enlargement of the liver, nor any very great diminution of the quantity of bile in the stools, although the patient's skin and urine are both distinctly impregnated with bile-pigment. Under these circumstances, only a partial suppression of the biliary secretion can be said to have occurred. Nevertheless the case may be a very dangerous one, for the constitutional disturbance may be very great. So great, indeed, as to induce a speedy fatal termination. More than once have I seen this happen in cases where the usual medical attendant, as well as myself, has been for a time at least thrown off guard.

As an example of the truth of this remark, I shall briefly cite a case which I saw along with Dr. Duke of Sydenham, who, at the time, furnished me with the following brief history of the case, when neither he

nor I had the slightest intention of putting it into print; and consequently, though imperfect, it is none the less valuable as a clinical history of the case.

'Captain P. D., Royal Navy, aged 46. Left the navy five years ago. Just before leaving the navy, he was on the Mediterranean station three years, during which time he had a great many attacks of fever. After leaving the navy in 1873, he went to Sierra Leone, where he stopped three months, during which time he was three times down with fever and ague—something of the same type as he had in the Mediterranean. He reached home in March 1875, much reduced by fever, which he first got in China in 1857. He went down to Norfolk, and gradually improved, and became quite strong. He came up to London, early in 1876, in good health, and during the rest of the year he was well. In 1876 he went before a Naval Medical Board, and was pronounced sound in health. At this time he was living at Penge. During 1876 saw a doctor, occasionally, for gout. In October 1876 came to Sydenham, and was then in good health. Walked, at Easter time, thirty miles on two consecutive days. Early in 1878 I first saw Captain D., professionally, with an attack of jaundice, which *was not* preceded by pain, and, at that time, no fever. He felt very weak at this time, and had a good deal of mental anxiety. After being under my treatment two months, and the

jaundice not going, Captain D. consulted Sir J. Ferguson, who told him that his liver was enlarged, and that the medicines he was taking were suitable, but that he might expect to be jaundiced two or three times before he got well. A month after this, Sir J. Ferguson saw him again, and said the liver was now normal, that it was inclined to be hard, and he would alter the prescription, so as to prevent its getting too small. In July of this year he was at Plymouth for a fortnight, when he had a slight attack of ague, which he has had more or less ever since. The first severe attack of an aguish character took place about six months ago (Nov. 29, 1878).' .

Many persons would have considered this patient to be in no great danger. Yet, from my knowing the insidious history of these cases, a somewhat dubious prognosis was arrived at, which the sequel, unfortunately, but too well verified; for the poor patient died, under the care of Mr. Douglas Duke, at Hastings, where we sent him to winter, within four months from the date of our consultation. The fatal termination being hastened by hæmorrhage from the bowels—a not uncommon occurrence when the liver becomes atrophied after prolonged malarial enlargement.

Malarial hepatitis, both in its acute, subacute, and chronic forms, frequently ends by inducing suppuration of the tissues of the liver. I believe I have

seen cases where hepatic abscesses have formed twenty years after the patients have returned to England invalided on account of jungle or other malarious fevers. In fact, the poison of the worst forms of malaria seems to saturate the tissues and adhere to the constitution with as much tenacity as the poison of syphilis. For there is no period of an individual's life, after he has had a bad attack of malaria, at which he may be said to have completely got over it. While in the act of revising this (September 23, 1879), I have a marked example of the kind in my mind's eye. For, two days ago, I was summoned to meet Mr. R. Phillips, of Leinster Square, in consultation regarding the case of a gentleman, aged 70, an old Indian who had returned to England nearly twenty years ago after having been saturated with jungle-fever and other malarial poisons, from the effects of which he is still suffering. On my arrival at the house, I found him in a distinctly marked aguish rigor; and on making a physical examination of his liver I found it greatly enlarged, both perpendicularly and laterally. The dulness in the perpendicular nipple line was over six inches, and Mr. Phillips informed me that it was then small in proportion to what it had been four weeks previously, when, he said, it had reached below the umbilicus. In this case, the history, as well as the physical sign of intense pain on pressure

over a circumscribed space at the centre of the lower margin of the liver, with a corresponding feeling of fulness, led to the diagnosis of a small chronic abscess; and this, too, notwithstanding that the patient had been away from India, and out of all malarial miasma, for a period of nearly twenty years. The history of the case precluding the probability of the suppurative hepatitis having originated earlier than a few months before I saw him. Moreover, I ascertained from Mr. Phillips that this gentleman had, while in India, suffered not only from jungle-fever and true aguish attacks, but from dysentery in all its worst forms.

I have not yet done with the malarial kinds of hepatic disease; for malaria, both of an aguish and a febrile variety, has much more to do with all the various forms of tropical liver congestion than the mere factor of heat. I question, indeed, if mere heat, *per se*, gives origin to hepatic congestions, for hundreds of English men and women residing in intensely hot but otherwise salubrious places are known not to be troubled with liver affections in a greater ratio than their compatriots dwelling in temperate zones. Moreover, the fact of ipecacuanha and quinine being the sheet-anchors in the treatment of tropical hepatic congestions likewise goes to the support of the theory that it is miasma, not heat, which is their exciting cause.

Malarial poisons not only produce hepatic, but even also renal congestions; and the two in conjunction produce an as yet mysterious chain of characteristic, though anomalous, symptoms, which I first brought under the notice of the profession in my monograph on jaundice published twenty years ago, and subsequently, in a more developed form, in a paper read on May 9, 1865, before the Medical and Chirurgical Society, and published in the forty-eighth volume of its 'Transactions,' under the title of 'Intermittent Hæmaturia,' but which, now that I know a great deal more of the matter, I believe I ought rather to have called

Paroxysmal Congestive Hepatic Hæmaturia.

The most remarkable features of this affection consist in the strange fact that although the abnormal urine passed by the patient during the attacks contains the whole of the ingredients of the red blood-corpuscles, scarcely a single entire blood-cell is to be detected in it by the microscope. Their *débris* being at the same time visible in every direction. Although, as I have already shown, and shall still further show, the urine is nearly always more or less abnormal in all cases of hepatic disease, in no single form of liver, or any other disease, indeed, is it so curiously abnormal as it is in this paroxysmal congestive hepatic hæmaturia. More-

over, in this remarkable affection, though highly albuminous, the urine possesses the notable characteristic of combining a high specific gravity with a great coagulability. While in renal albuminuria the specific gravity is always under 1010, in this form of hepatic albuminuria it is almost invariably over 1015.

No more correct idea can be given of one of the special features in this peculiar hepato-renal affection, than by quoting the reply of one of the patients suffering from it, when asked what was the matter with him. His answer was given to me in these words—'I can't tell you; but each time I get cold hands or cold feet I pass bloody urine, while my urine is at other times perfectly healthy.'

In the other case, which I am about also to relate, the urinary symptom was not traceable so much to the effects of cold as to malarial poisoning; and as it was the one which first fell under my observation, I shall cite it first.

Dr. ——, a member of our own profession, after several years' residence in one of the West Indian Islands, was, in consequence of repeated attacks of intermittent fever, forced to give up practice and return to England, where for the first two years he was still liable to occasional outbursts of his old enemy. On one occasion, while consulting me regarding his case, he mentioned what he considered

to be a very peculiar symptom, namely, that he occasionally suddenly passed five or six ounces of urine of a dark red or chocolate colour, a symptom which would recur once in twenty-four hours during two or three days, and then as suddenly disappear. Never having before met with such a case, I requested him to send to me, on the next occasion, a specimen of the fluid. In the succeeding November (1861) I received from this gentleman three samples of urine —one passed at 8 a.m., which was clear, pale, of a specific gravity of 1025, of an acid reaction, depositing no lithates, and containing no albumen, being, in fact, normal in every respect. Another quantity, passed at 2 p.m. of the same day, of a dark chocolate-brown colour, opaque, turbid, having a specific gravity of 1032, of an acid reaction, depositing lithates, containing a large quantity of albumen, some sugar, and a large excess of urea (3·6 per cent.). The deposit from this specimen of urine, when examined with the microscope, was found to contain nucleated epithelium (fig. 9, p. 380), some granular cells, and a large quantity of free granules of a brownish-red hæmatin colour, scattered among which were a considerable number of renal tube-casts. The tube-casts presented one or two remarkable peculiarities, namely, that the majority of them were short and broad, and filled chokefull with brown pigment, as represented in

the woodcut. Besides these, there were a small number of fine, long, pale tube-casts, with only a few granules of dark pigment distributed in them ; these looked not at all unlike the renal tubes emptied of their epithelium obtained by scraping a section of fresh kidney. No blood-corpuscles were to be found in this specimen of urine. The third sample of urine sent by the gentleman in question was passed in the evening of the same day, and presented a striking contrast to that just described. It was normal in colour, contained no albumen, deposited a small quantity of ordinary coloured lithates, among which were neither tube-casts nor granular cells. The specific gravity of the liquid was 1021. Its reaction was acid, and its percentage of urea exactly half (namely, 1·8 per cent.) of that of the preceding specimen. These three different conditions of the urine were certainly very peculiar ; for had the morning's specimen alone been brought under the notice of the physician, he could never have dreamt of the existence of any urinary affection. On the other hand, if the single specimen of urine passed four hours later had been submitted to his inspection, he must have come at once to the conclusion that there existed very grave organic changes in the renal organs. Whereas neither the one nor the other of these opinions, as I shall presently show, could possibly be correct.

The gentleman alluded to, at the time when he passed these urines, was labouring under hepatic derangement; being, in fact, slightly jaundiced, as a result, most probably, of the malarial poison, from the effects of which, as before said, he had not yet entirely recovered.

The varying conditions of the three urines clearly pointed to intense congestion of the chylopoietic viscera of a transient and periodic character. Suiting the practice to the theory, mercurials, and afterwards quinine, were taken by this gentleman, in order to remove the congestion of the chylopoietic viscera, and check the periodicity of the disease. The results were most favourable, for, although twenty-one years have passed away since then, he has never had a recurrence of these urinary symptoms, and is now alive, well, and in active practice in Devonshire. I shall now proceed to call attention to the second case, which is equally traceable to malarial liver disease, notwithstanding that the man was never out of England.

On December 16, 1864, M. N., a dark, sallow-complexioned, careworn-looking man, was sent to me by my colleague, Professor Fox, in consequence of his case presenting unusual characters.

The history of the patient is briefly as follows:—He is a blacksmith by trade, thirty-two years of age, and unmarried. Until two years ago, he considered

himself perfectly healthy, having always been able to do forge work without either difficulty or inconvenience, having, in fact, been a strong man.

Two years ago, he, for the first time, observed that he occasionally passed urine as dark as brown old ale, while that voided at the preceding and succeeding micturitions possessed the normal colour and transparency.

Twelve months later—a year before I saw him—the urine for the first time assumed the colour of blood—a symptom which greatly alarmed him, as it recurred about three times a week during the whole of that winter, except during a fortnight in January, while working in the open air, when it became still more frequent, occurring about once every day. Sometimes the attack of bloody urine lasted over two micturitions, amounting to a period of from four to five hours. In the spring of that year, as the warm weather advanced, the attacks gradually became less frequent, until from the month of May to September they entirely ceased. In September, however, they reappeared at intervals of about every ten days, the intervals gradually diminishing, until a fortnight before he came under my care, when he passed bloody urine every other day; and for the last five days he had passed it every day at irregular hours varying between 10 a.m. and 6 p.m. The quantity usually emitted was about six ounces. The

patient further stated that since the commencement of his illness, with the exception of the summer interval, he was constantly under treatment for bloody urine at different London hospitals without receiving the slightest benefit.

On his first visit to me, the man brought with him two bottles containing the urine that he passed at 9 a.m. and at 2 p.m. on the previous day. The former sample was clear, transparent, straw-coloured, and normal-looking ; the latter, a dark purple blood-coloured fluid. On carefully cross-questioning the patient as to the origin of these liquids, he stated that the dark urine was usually passed about an hour after his feeling cold ; that the urine did not invariably become clear at the next micturition ; and that occasionally it did not resume its perfectly natural colour until he had emptied his bladder three times. He stated, moreover, that he then felt cold, and that even during the time he was kept in the waiting-room he had passed four ounces perfectly similar to the bloody-looking fluid which he had brought with him. He was accordingly requested to go behind the screen and make some more, which he immediately did, and produced about two ounces of a liquid of a dark purple-red colour. As he complained of feeling intensely cold, notwithstanding that he was sitting in front of a large fire, the temperature of the palms of his hands was taken, and

found to be only 60° Fahr., while the temperature of my own hand was 95·4° Fahr. The temperature of the patient's axilla was also carefully taken, with as little disturbance to his dress as was possible, and, in spite of his being well clad with warm clothing, it was ascertained to be only 96·1° Fahr., a result which entirely confirmed his statement regarding his sensations of cold. He moreover added that he was a Londoner, and had never, as far as he knew, suffered from ague; the most that could be ascertained on this point being that on some occasions he had felt so cold as to shiver during the night, which shivering was not, however, followed by a true hot stage.

As before mentioned, the man was dark complexioned, and had a sallow look. Which sallowness appeared to be due to disturbance of the hepatic functions. He admitted that he was a very bilious subject, but denied having ever had any hepatic affection beyond what might be included in the term functional derangement, and this had never at any time amounted to actual jaundice.

It will be observed that this and the preceding case present many features in common, the only apparent difference being that while the first could be distinctly traced to tropical malarial poison, the second appears to be simply the result of the direct effects of cold and damp acting upon a predisposed

constitution. Such, at least, was the theory I formed of the disease at the time, and, accordingly, the line of treatment recommended in ordinary hæmaturia was abandoned, and the plan of treating it as the result of malaria adopted, as in the first case. A course of treatment which proved most beneficial, for before twenty-four hours had elapsed the disease received a check, and by the end of forty-eight hours it may be said to have completely disappeared, for from that time he never had a single recurrence of his urinary symptoms. The patient was a regular attendant at the hospital during the whole winter months, coming once a week, no matter how cold or wet the day was, up to the time the warm weather set in, when, by permission, he ceased his visits.

The amelioration of the condition of the patient in this case can scarcely, I think, be attributed to anything else than the effect of the treatment, as the diet and other conditions under which he was placed remained entirely unchanged. We cannot even suppose that the weather had anything to do with it, for the temperature of the atmosphere in the last two weeks of December, throughout January, February, and the beginning of March, was often lower than in any of the preceding months of the winter. The patient was at no time taken into the hospital, but made to come, as already said, once a week, no matter whether the day was wet or dry,

cold or warm. Moreover, until the very day on which the treatment was commenced, the patient's condition had been gradually becoming worse and worse, while, as just said, within twenty-four hours after it was begun, the disease had evidently received a check, and within forty-eight, the urinary symptoms had entirely disappeared.

At page 756, vol. i. (3rd edition), of Sir T. Watson's 'Lectures,' it is mentioned that when quinine, given alone, fails to cure an ague, a few grains of calomel, followed up with quinine, will often entirely check the disease—a fact which rather goes to support the view that even the second case might be due to a form of malarial poisoning.

These remarks might be allowed to end here; but as I consider that the pathology of such cases as have just been described is of great clinical value in connection with hepatic disease of a malarial character, I shall say a few words more regarding the condition of the urine, as by so doing it will not only be seen on what grounds I founded my diagnosis, but I may perhaps aid the labours of some future inquirer, who may have the good fortune to throw more light upon the nature of these cases than the data I have at present at command enable me to do.

The man was ordered to preserve all the urine he passed during the forty-eight hours after I first

saw him, and to put what was passed at each micturition into separate bottles.

On examination, it was found that the specimen passed at 8.30 a.m. was normal in colour, devoid of any sediment, six and a half ounces in quantity, acid in reaction, and of a specific gravity of 1010; it contained 1·75 per cent. of urea, traces of sugar, but no albumen.

That passed at 2 p.m. was dark red, almost black-looking, six ounces in quantity, acid in reaction, of a

FIG. 9.

1. Granular tube-casts.
2. Large granular cells.
3. Mucus-corpuscles.
4. Free hæmatin.
5. Oxalate-of-lime crystals.
6. Amorphous urates.

specific gravity of 1017, and, on standing, deposited a copious precipitate of dark-coloured urate of soda, leaving the supernatant liquid quite clear, and of a fine rich port-wine colour. This urine contained 2·5 per cent. of urea, was highly coagulable by heat and nitric acid, and gave evidences of traces of sugar. On examining the deposit from it under the microscope, although scarcely a single blood-corpuscle was

to be found, it contained a great abundance of granular tube-casts (fig. 9, 1), large granular cells (2), free granular matter of a hæmatin colour (4), and among the urates a few octahedral crystals of oxalate of lime (5). It will be seen that this urine presents a striking resemblance, both in its chemical and microscopical character, to that of Dr. ——, being of high specific gravity, coagulable by heat and nitric acid; containing a large excess of urea, abundance of granular tube-casts, and scarcely any blood-corpuscles. For all practical purposes one may say that there were no blood-corpuscles in the urine, for out of sixteen of the gentlemen attending my practical class only two found any, and even then there were only one or two in the field of the microscope; while, had the case been one of ordinary hæmaturia, the absence of blood-corpuscles would have been the exception, not the rule.

I was particularly struck with the resemblance this urine bore to the urine I have occasionally seen dogs pass after I had injected either bile or bile-acids in toxic doses under the skin of their backs. Their urine not only occasionally presented exactly the same colour, but contained lots of granular tube-casts, and still further closely resembled this human urine in being coagulable by heat and nitric acid. All this leads me to the conclusion that the condition of the urine in cases of paroxysmal hepatic albu-

minuria is in greatest part due to disorder of the biliary secretion brought about by the direct result of malaria acting upon the liver.

The urine passed at 6.30 p.m. was five and a half ounces in quantity, slightly smoky in colour, with a moderate deposit of pale urate of soda, a specific gravity of 1016, and acid reaction; it contained 2 per cent. of urea, and was slightly coagulable by heat and nitric acid. The deposit, when examined by the microscope, was found to contain only a few granular tube-casts, one unusually long one, with some granules in it; a few mucus-cells, no crystals of oxalate of lime, no blood-corpuscles, and only small collections of pigment scattered among the amorphous urate of soda.

The urine passed at 9 p.m. was about six ounces in quantity, perfectly normal in colour, without any sediment; had a specific gravity of 1016; acid reaction; contained 1·84 per cent. of urea, and not a trace of albumen.

The urines passed on the following day were all normal in colour except one, namely, that passed at 10 a.m., which was of a slight brownish-red tint; it deposited a copious sediment, coagulated freely on the application of heat and nitric acid, and contained 2·05 per cent. of urea. In the sediment of this urine were found a number of granular tube-casts, but nothing like the quantity in that of the well-marked

specimen. This specimen presented, in fact, the last appearance of the disease, for from then until he left the urine never again became either albuminous or of a dark colour, and only twice, on the 12th and 20th of January, did it deposit any precipitate. On the first occasion the patient suspected a recurrence of his old disease, a suspicion which, fortunately for him, was not verified, for, on testing the urine, it was found to become perfectly transparent on the application of heat, and to be entirely devoid of albumen or tube-casts, the turbidity being simply due to the presence of urate of soda. The second specimen was exactly similar.

There are one or two points regarding the condition of these various urines requiring special notice.

1. The presence of the granular tube-casts clearly points to congestion of the renal organs, but their appearing and disappearing in the course of a few hours at the same time proves that it was not an ordinary case of renal congestion.

2. The almost total absence of blood-corpuscles, notwithstanding the hæmorrhagic appearance of the urine, stamps the case as being entirely different from ordinary hæmaturia, and shows its resemblance, in this particular, to that variety of non-intermittent urohæmaturia in which the contents of the blood-corpuscles alone pass into the urine.

3. It cannot be regarded as a sample of simple intermitting albuminuria; for although the protein substance coagulable by heat and nitric acid has been hitherto spoken of as albumen, it differed very materially from the albumen of blood-serum in its ready solubility in an excess of acid. In making the analysis it was found necessary to be exceedingly careful with regard to the amount of nitric or acetic acid employed, for after the coagulable point was arrived at the addition of a single drop more of either of these acids instantly redissolved the coagulum, and set the colouring matter free, a circumstance which, taken in conjunction with the uniform diffusion of the pigment and the coagulable matter in the liquid, leads to the conclusion that it was not simply the albumen of the blood-serum, but the hæmato-globin itself, which was excreted by the kidneys.

4. The case was not one of urohæmaturia, such as I have elsewhere described,[1] for two reasons—first, on account of the urine being coagulable by heat and nitric acid; and, secondly, from the fact of the addition of acids diminishing instead of increasing the dark tint of the urine.

5. The bilious appearance of the patients in both cases would lead to the belief that the attacks were

[1] *Lectures on the Urine, and Diseases of the Urinary Organs*, Churchill & Co.

connected with the disturbance of the hepatic function, which, in one case at least, was distinctly traceable to malarial poisoning.

6. As the bile-acids have a powerful disintegrating effect on the cell-walls of the red blood-corpuscles, it has once or twice crossed my mind that this peculiar condition of the urine in paroxysmal hepatic hæmaturia may possibly be due to an abnormal quantity of bile-acids in the circulation. Although this is only a passing idea, it may nevertheless be well worthy the consideration of future observers who may have the opportunity of studying cases of this kind, and hence my reason for here directing attention to it.

7. The copious deposition of urates, as well as the excessive elimination of urea, which takes place during the attack, points to considerable general constitutional disturbance.

Lastly, the transitory condition of the urinary symptoms shows that, whatever might be the nature of the disease, the exciting cause could not be in constant operation, unless we admit that it required distinct periods of incubation as in ague, which we know is liable to assume an almost endless variety of forms. In conclusion, I have only to remark that it is of great clinical importance to be able to make a correct differential diagnosis between cases like the present and those of ordinary hæmaturia, for without

it, it will be utterly impossible to treat them with any chance of success. The last-quoted case is, indeed, a striking illustration of the truth of this remark ; for, notwithstanding the patient having been at different London hospitals, under the care of men of high professional standing, he failed to obtain relief, in consequence of the orthodox line of treatment laid down for ordinary hæmaturia having been adopted, and, as has been said, was at once cured when the case was treated as one resulting from the combined effects of hepatic and renal malarial disturbance. Since these cases were first published I have had at least a dozen of a precisely similar character under treatment, all of which made speedy recoveries.

I cannot refrain from here remarking that since I first called attention to this peculiar affection in 1863, a whole host of cases have been reported by writers both at home and abroad—but principally at home—and that the majority of them have not only manufactured new names for the disease, according to their own theories regarding its pathology, but that some of them have not had even the common politeness to mention so much as my name in connection with it, although they have reiterated almost my very words regarding the peculiar characters of its urine, by which they would lead—no doubt unintentionally—the uninitiated to suppose that they

had discovered them all for themselves. Dr. Jones of Louisiana has called attention to the fact that in his district there is a very marked acute form of this hepato-renal malarial affection, which, he says, is characterised by well-marked jaundice as well as hæmaturia.

'In some cases immense quantities of green biliary fluid, or liquid tinged with bile, are vomited, and the patients die in a state of collapse, with blue-mottled, purplish extremities, and sunken, pinched features. As a general rule, suppression of the functions of the kidneys is a fatal sign, and, as in yellow fever, is sometimes attended with convulsions, coma, and delirium. And whilst some of the symptoms—as the nausea, incessant vomiting (in extreme cases black vomit), deep jaundice, and the impeded capillary circulation—resemble those of yellow fever, yet there are marked differences between this disease and yellow fever.

'The pathological changes observed after death are characteristic of paroxysmal malarial fever, and not of yellow fever—viz., enlarged slate- and bronze-coloured liver, loaded with dark pigment granules, deposited in greatest numbers in the portal capillary network; gall-bladder distended with thick, ropy bile, presenting, when seen *en masse*, a greenish-black colour, and in thin layers a deep yellow. As much as 1,000 grains of bile of high specific

gravity has been obtained from the gall-bladder, whilst in yellow fever not more than 120 grains of bile are, as a general rule, contained in the gall-bladder.'

This form of jaundice, he says, prevails only in certain years, and appears to be dependent to a large extent upon the degree of heat and moisture, as well as upon the amount of organic matter in the soil.

Treatment.

As regards the treatment of cases of malarial hepatitis and jaundice, whether in a smart acute or a severely chronic form, the free use of mercurials, in combination with quinine, salicine, or ipecacuanha, is almost invariably absolutely essential in order to effect a complete cure. But, sad to say, even under the most favourable conditions, both of hygiene and treatment, many of the cases that have been long subjected to some of the worst forms of malarial influence succumb, from the constitution having become, as it were, thoroughly and irretrievably undermined by the poison.

After the preceding descriptions of the etiology, symptoms, and pathological appearances, in the two kinds of the so-called yellow fevers, I think I need offer no further apology for recommending the total abolition of the name of 'yellow fevers,' and substituting, as I have done, that of 'contagious and mala-

rial jaundice.' For I presume that even the most nonchalant of my readers must have observed for themselves that not only are the so-called yellow fevers nothing but different forms of jaundice, but that, though allied to each other in some of their symptoms, their pathology—at least in so far as regards their origin and cause—must be looked upon as being entirely different. In so much as the one —specific yellow fever—as is proved by its contagious character, is due to the introduction into the system of a peculiar and special epizootic poison. Which not only germinates and multiplies within the animal organism, but, like smallpox or scarlatinal poisons, can be communicated from an infected to a healthy individual. Whereas the other form of so-called yellow fever—*febris icterodes remittens*—malarial jaundice, is, in its turn, as palpably due to the introduction into the system of a malarial or paludal miasmatic poison. Which can only be obtained by the human body directly from the atmosphere of the districts in which it is indigenous, and therefore cannot be propagated by contagion from one human being to another.

Acute Atrophy of the Liver.

Acute, or, as it is sometimes called, yellow atrophy of the liver—in consequence of the yellow appearance of the hepatic tissue—though a disease of temperate

zones, I unhesitatingly assert is a mere sporadic form of the contagious jaundice of the tropics. At this conclusion I have arrived—

1. On account of their symptoms as well as their signs being the same.

2. The *post-mortem* appearances in both being identical.

3. On account of the similarity existing between them in point of fatality.

4. Because, as is now known, acute atrophy of the liver may, even in temperate zones, manifest a contagious character.

Before more fully alluding to the points of identity in these two forms of disease, I shall go fully into the signs and symptoms of acute atrophy; for the account I shall give of its clinical history will very materially facilitate the understanding my exposition of the identity of these two forms of disease, whose pathology has so long been a stumbling-block to practical medicine. For on account of the one being only indigenous and epidemic in certain tropical regions, and the other sporadic and never assuming a true epidemic form in temperate climates, it was thought that the pathology of the diseases must of necessity be different. I shall immediately show, however, that acute atrophy of the liver is often as sudden in its onset, as rapid in its course, and as fatal in its termination, as the most

virulent form of contagious jaundice. Added to which, that it manifests the same kind of cerebral symptoms, as well as identically the same kind of black vomit and tarry dejections (hæmorrhage from the digestive canal—stomach, and intestines). While similar kinds of urinary derangements are likewise its invariable accompaniments.

Like all forms of virulent disease—cholera, plague, malignant typhus, &c.—acute atrophy of the liver, contrary to what was until within the last few years supposed, attacks persons of either sex and at all ages. But it shows a predilection for females between the ages of fourteen and twenty-three, as well as for the puerperal state, especially the first four months of pregnancy. In this respect, again, it closely resembles Asiatic cholera and plague, which have a striking predilection for pregnant women.

This proneness of acute atrophy of the liver to attack pregnant women must not be allowed to lead one into the error of confounding the jaundice which it gives rise to with what is called the 'jaundice of pregnancy'—either epidemic or sporadic—which has an entirely different pathology, and is fortunately of a much milder nature. So much so, indeed, that Gooch, in his treatise on the diseases of women (p. 56, Sydenham Society's edition), is able to relate a case in which it recurred in the same patient during three successive pregnancies. Whereas a

pregnant woman, once the victim of an attack of jaundice the result of acute atrophy, is not likely to live to have another. But even when she has surmounted the disease, I know of no case on record where it has attacked her a second time. In this respect it resembles all other epizootic germ diseases in showing but little tendency to recurrence.

Predisposition is, in this as in all other cases of germ disease, necessary for its appearance. The urine becomes scanty, and of a bright saffron colour—quite different from the brown ale tint of ordinary jaundice urine. The stools become loose. The dejections are black and tarry, leading to the idea that they are loaded with bile. While the dark colour, on the contrary, is due to the presence of blood. In those cases where no hæmorrhage has taken place, the stools are of a pale colour. Sometimes, indeed, the patient passes no stool at all for several hours. A few hours later, extravasations of blood take place under the skin ; while hæmorrhages from the bowels, nose, and vagina, are frequently observed to occur. Lastly, delirium or coma generally closes the scene, within a couple of days, or at most a week, after the commencement of the violent symptoms. Frerichs, who has well described these cases, even says that 'in the severest forms the disease may run its course and end fatally within twenty-four hours.' [1]

[1] *Clinical Treatises on Diseases of the Liver* vol. i. p. 197.

As regards the morbid anatomy, little need be here said, as it may all be embodied by simply comparing it to what I have pointed out as occurring in the worst forms of contagious tropical jaundice. For it is invariably found that the liver is not only softened, but completely degenerated. Sometimes almost reduced to a yellow pulpy mass, and not one half of its normal weight. So that, like tropical jaundice, it is well described as one of the most formidable of human diseases, seeing that all these changes may occur in the brief space of a few hours.

In 1868, Dr. Grainger Stewart called attention to the fact that acute atrophy of the kidneys may not only be associated with, but precede, acute atrophy of the liver.[1]

It is generally asserted—and I repeat the statement, though I do not believe it—that mental depression is its almost invariable exciting cause. It may be a predisposing, but that it is an exciting cause I am inclined to doubt. In the two typical cases I am about to relate, the depression of spirits was manifestly the result, not the cause, of the attack. So I am inclined to think that the same is the case in the majority of others where marked depression of spirits is observed. It appears to me to be much more probable that the exciting cause is in all cases the direct accidental admission into the system of epizootic

[1] *On Bright's Disease*, p. 159.

disease-germs. To which it has in some few instances been clearly traced.

Symptoms.

The symptoms in an ordinary case of acute atrophy of the liver may be said to begin with a feeling of restless uneasiness accompanied with a gradually deepening yellow tinging of the skin, followed by headache, gastric derangement—vomiting—and pyrexia. In a few hours all these symptoms become alarmingly exaggerated. Bloody, bilious-looking vomiting sets in. The tongue becomes brown and dry. Shooting pains are complained of throughout the body. The patient assumes a depressed, typhoid look. The pupils become dilated. The mind wanders; delirium supervenes, coma follows, and the patient sinks and dies without a single break having occurred in the rapid downward progress of the disease. The speedy diminution of the hepatic dulness in the perpendicular nipple line is the most characteristic, as well as the most curious, physical feature in the case. From four, or four and a half inches in extent, the liver dulness may in the course of twenty-four hours diminish to two inches, to one inch, or even less. While before another twenty-four hours have passed away, its boundaries may be actually inappreciable on even the most careful percussion. While, after death, instead of the organ

weighing about fifty ounces, as it ought to do, it may weigh but little more than twenty or even fifteen ounces, and present no appearance of a liver at all, but merely look a soft, pulpy, structureless yellow mass. Another marked and curious feature of this incomprehensible form of disease—which so closely resembles in many respects the contagious jaundice of the tropics—is, that although there are marked jaundice and deep saffron-coloured urine, the stools, as in contagious jaundice, occasionally present all the appearances of bile about them—when none is actually present—from their containing blood, from the very beginning to the very end of the attack.

Treatment.

As can easily be imagined, in rapid cases of acute atrophy of the liver, one can often do very little in the way of treatment. For, treat them as one may, the majority of the rapid cases prove fatal. So the only advice that can be given with advantage is to say: Direct all your energies to thwart what appears to be the immediate cause of impending death. If there be violent vomiting or diarrhœa, try to stop it. If there be obstinate constipation, give an active alkaline purgative. If there be acute delirium, give antimony, and apply ice to the head. If there be hæmorrhage from mouth, nose, stomach, bowels,

or vagina, try to arrest it by the most appropriate ordinary means. If there be (as sometimes happens) a diminished secretion of urine, give non-stimulating diuretics. If there be hepatic pain, apply hot fomentations; and, above all things, support the strength of the patient by nourishing, non-stimulating, fluid diet. Avoid all lowering remedies.

At one time it was generally believed that all cases of acute atrophy of the liver were necessarily fatal. Fortunately this is not the case, for in some the violent symptoms gradually disappear, and, as in contagious jaundice (yellow fever), recovery takes place after free evacuation of the bowels.

I shall now relate two cases as an encouragement to others never to despair of a patient's recovery, even in very bad cases. For I believe, so long as life exists, there may yet be hope. I also select them as being examples of the disease occurring not only in the opposite sexes, but at totally different periods of life. The first I shall relate is that of a gentleman aged 58 (brought to me by Mr. Roberts—formerly of St. Asaph, now of Denbigh), in whose case I took more than ordinary interest, from his being the son-in-law of one of our former presidents of the College of Physicians. He began to feel ill a fortnight before I saw him, with loss of appetite, sickness, vomiting, and sallow-

ness of the skin, but no pain. On June 9, 1869, when I first saw him, within a few hours after his arrival in London, his appearance made a profound impression on my mind. He had become dangerously exhausted in the train on his way up to town, and had I been asked what I thought of his chances of recovery, I should have estimated the value of his life at not more than a few hours' purchase. He was deeply jaundiced. He had a typhoid, dark-furred tongue. The skin was burning hot, though moist and clammy to the touch. The pulse rapid, thready, weak, and intermitting. The expression dull and heavy. The intelligence sluggish, and his manner restless and fidgety. He complained of intense headache and general *malaise*, but no hepatic pain whatever, not even on firm pressure. On percussion, the hepatic dulness in the perpendicular nipple line was exceedingly difficult to ascertain in consequence of its small extent, coupled with the thickness of the abdominal parietes—for the patient was rather stout; but in the nipple line it was certainly not more than one inch in extent. While in the axillary line the perpendicular dulness was estimated at only one and a quarter inches. The urine was so loaded with bile as to be almost black. There was hæmorrhagic vomiting and bloody stools, but no distinct diarrhœa. The case was at once seen to be one of acute or yellow atrophy; and a more

unfavourable one in appearance could scarcely be imagined. Yet, strange to say, the bad symptoms all yielded to treatment with marvellous rapidity; and the patient, to the astonishment of both Mr. Roberts and myself, got speedily well; and what is still more surprising is, that his health has been satisfactory ever since. I have had occasion to examine the size of the liver of this gentleman several times during the last ten years (as he usually presents himself for inspection when he visits London), and from within about the first eighteen or twenty months after the attack until now the liver dulness in the perpendicular nipple line has never been less than four and a half inches. After the attack the liver slowly but gradually increased in size. It was three inches within ten months after the attack, and I think had reached its normal dimensions in about fifteen months from the time he was first seen by me in London.

Although my memory is a good one, in order to make perfectly sure that the above statements regarding the case were correct, I wrote to Mr. Roberts, and the following is his reply, dated Denbigh, September 23, 1879 :—

'R. A. A——, Esq., aged 58, was suddenly attacked with jaundice, and without any visible premonitory signs other than a general feeling of languor during the previous fortnight. Among the

first immediate symptoms were restlessness and fearful headache. He was easily agitated, and his limbs became more or less tremulous. The pulse was abnormally quick ; the tongue coated, and of a typhoid character. Another symptom was vomiting ; and on the morning of the third day the vomit was like coffee-grounds. [From blood acted on by gastric juice.—G. H.] The diminution in the hepatic dulness was very sudden and marked, and he complained of slight pain on pressure in the hepatic region. The urine passed was of normal quantity, but of the saffron colour peculiar to jaundice ; it had an acid reaction. The bowels were confined, and the fæces totally devoid of biliary matter, being of a doughy white clay colour. At my earnest request, on the fourth day, I got him up to London to consult you, with what result you can best describe. That was ten years ago ; and now to all appearance he may live other ten years. I may add that on the whole Mr. A——'s health has been very satisfactory ever since.'

The treatment I adopted in this case was very simple. Hot bath, hot fomentations to the liver, grey powder, rhubarb and magnesia. Along with a carefully regulated diet.

The other case, as I before said, I shall cite not only from among the opposite sex, but also at the opposite extreme of life, and in a state of pregnancy.

It has an additional importance attached to it from the fact of the disease having attacked the patient almost as soon as she was capable of becoming pregnant. The patient was a mere child in appearance, who at the early age of fourteen years and nine months was already three months advanced in pregnancy.

She was admitted into University College Hospital, under my care, on July 30, 1864, and gave the following history as abbreviated from the case-book.

Eliza N. Has always been subject to 'bilious attacks' and 'fainting fits.' The latter usually coming on in the morning, when getting up. She states that she has had all the diseases of childhood, as well as smallpox and typhus fever. Has not menstruated for three months.

On examining the skin of the chest in order to judge of the depth of the jaundiced tint, I was forcibly struck with the unusually dark colour of the nipples and their surrounding areolæ, which was all the more remarkable on account of the smallness of the mammæ. A patch of a dark chocolate tint of exactly the size of a half-crown in diameter, with unusually large papillæ upon it, surrounded each nipple, and this, from the fact of the girl's hair being fair, notwithstanding her childish appearance, and her age being only fourteen years and nine months, raised in my mind the suspicion that

she might be pregnant, and the jaundice be in some way or other traceable to the pregnancy. For the depressed and anxious look of the patient, coupled with the comparatively slight tint of the jaundice, had already raised the suspicion that the case was not one of ordinary jaundice.

Although the abdomen was not perceptibly enlarged to the naked eye, when the hand was placed in the umbilical region, immediately a well-defined moveable tumour was found, pear-shaped and somewhat bigger than a fœtal head. On auscultation a fœtal heart was distinctly heard beating 160 per minute, and in the left iliac region a placental murmur. She had had no morning sickness. On being closely questioned, patient acknowledged the possibility of her being pregnant, and said she thought that she might have become so on May 13, and that it was impossible to have been sooner, as she had just recovered from an attack of small-pox which had confined her to bed for three weeks. So, if her version were true, she was only two months and three weeks pregnant. The case-book further says :—

Present attack: came on three weeks ago, with pain over her eyebrows. She at the same time noticed that her eyes became yellow. The night before she had a sharp pain coming on suddenly, and continuing the whole of that night in hepatic region ; has slight pain there now on pressure. No pain elsewhere.

States that everything she takes makes her sick. Bowels during the last three weeks have been much relaxed, and stools very light-coloured.

Skin is of a slightly greenish lemon yellow; the conjunctivæ of a more marked jaundice tinge. Complains of pain in the epigastric region after eating, increased on firm pressure. Unusually marked tympanites in hepatic region. Hepatic dulness in a line perpendicular to nipple, is very difficult to make out, but may be put down at only an inch in depth, commencing at one inch below the nipple.

Urine high-coloured and stains the linen of an ochre colour.

On August 10 the jaundiced tint of the skin had much decreased. While the hepatic dulness had increased to an inch and a half in depth. The stools were darker in colour, and the urine much paler. One thing about it was, however, peculiar—namely, that although the patient was taking three-grain doses of benzoic acid three times a day, not a trace of hippuric acid could be detected in the urine. On the 24th the patient was so much better as to be dismissed.

Through the kindness of Dr. Wilks I had the opportunity of examining the liver, and analysing the urine, in a typical fatal case of acute atrophy, which he reported in the Pathological Society's 'Transactions,' vol. xiii. p. 107. The brief history of the case

is as follows :—E. K., aged seventeen, a married woman, in the third month of pregnancy, was seized with a bilious attack, and jaundice, after having a violent quarrel with her husband, who accused her of infidelity. The patient was first under the care of Mr. Bisshopp, of South Lambeth, who found her suffering from jaundice, accompanied by some febrile symptoms, and vomiting. In two days she became delirious, had violent screaming, and convulsive fits, which were rapidly followed by unconsciousness. Next day the patient was seen by Dr. Wilks ; she was then quite insensible, with slight stertorous breathing, and foam on the lips. The pupils were moderately dilated, and sensible to light. The pulse 120. The hepatic dulness reduced to a narrow band over the lower ribs. No urine had passed for twenty-four hours ; a catheter was therefore introduced, and twelve ounces of clear bilious-looking fluid were drawn off. This urine I had the opportunity of analysing a few days afterwards. It was then of a yellow-ochre colour, and contained a considerable deposit. The analysis of this urine is given in detail at page 746, where I call special attention to the diagnostic value of tyrosin and leucin, two chemical products which are never absent from the urine in cases of acute atrophy of the liver.

During the night before her death the patient aborted, and lost a considerable quantity of blood by

the vagina. The whole duration of the disease was merely six days, and the more urgent symptoms only manifested themselves two days before the fatal termination.

After death the liver was found to be very small in size, not exceeding, as was supposed, 1½ pound in weight. It was deeply stained yellow, and its cells were found to be small and broken up ; not an entire cell could be detected by either Dr. Wilks or myself —nothing, indeed, but a quantity of *débris* of hepatic tissue, and fat. The gall-bladder was contracted, and contained only a little mucus ; the urinary bladder was empty.

In a case of acute atrophy of the liver, which was brought to the London Hospital, and placed under the care of Dr. Head, although the patient was a woman aged twenty-eight and of average size, the liver at the necropsy was found so small as, in the words of the reporter, ' not to have been seen on cutting through and folding back the abdominal walls ; until, on drawing down the coils of intestine, it was observed shrunken, as it were, and lying up under the ribs against the diaphragm. When removed, it was seen to be smaller and much thinner than natural. It was very flaccid, and folded by its own weight over the hand. It weighed 1 lb. 15 oz. Its surface was smooth, and of a pale reddish-yellow colour. On section, its substance for the most part had a Turkey-

rhubarb-like yellow appearance ; almost all signs of lobular structure were lost. Here and there, however, were portions that seemed more healthy ; in such parts the intralobular veins were distinct ; and here also there were some minute blood-extravasations. The gall-bladder was almost empty. The microscopic examination of the liver showed there was recognisable lobular arrangement ; and, although the minute biliary ducts seemed smaller than natural, yet their outline was distinct, but the liver cells were greatly altered, broken down, and almost completely disintegrated, and in their place there was a larger quantity of granular *débris*. There were many granules, which permitted light to pass readily through their centre ; so-called fat-granules ; also some yellow seemingly bile-pigment. The fibrous matrix of the liver was very distinct. The capsule was for the most part normal ; but from its under surface a number of corpuscles were seen extending into the liver-substance, looking as if some new growth were going on at the time of death. The fœtus, weighing 5 lbs. 14 oz., showed no signs of hepatic derangement. (' British Medical Journal,' July 25, 1874.)

In a case reported in the ' British Medical Journal,' 1871, p. 367, by Dr. Clements, the liver of a girl aged seventeen, who died of acute atrophy, weighed only thirteen ounces.

It is not in every case of acute atrophy of the liver

that the tissue of the organ is found soft and friable; for in a case on which I was required to make a report for the Pathological Society in 1864 along with Dr. Murchison, the organ which was removed by Dr. Robinson from a soldier in the Scots Fusilier Guards, aged 20, though of a deep yellow colour, was found to be quite dense in structure. The organ, instead of weighing 4 lbs. as it ought to have done in a man of his size and age, weighed only 2½ lbs. Yet, notwithstanding its dense structure, scarcely a single entire hepatic cell was to be found in it. Only granular matter and oil-globules. In the kidneys were found crystals of tyrosin; but none were noticed by either of us in the liver. The crystals found in the kidneys were spiculated balls and stellate needle-shaped groups. The clinical history of this man's case was somewhat peculiar. He was being treated in hospital by Dr. Robinson for soft chancre on the prepuce, when he was noticed to be slightly jaundiced; but his general health seemed to be unaffected. When on the morning of the second day after his admission (having taken a mercurial aperient the night before) he was found in a comatose state by the hospital-sergeant. The man was passing his urine involuntarily, and it was the fact of his comrade in the next bed noticing the urine trickling through the bed-clothes on to the floor that first called attention to the poor man's comatose condition. When

visited shortly afterwards by Dr. Robinson, his pupils were found dilated and insensible to light. His skin cold. His respiration laboured. His pulse weak and slow. Nothing else was noticed until later in the day when he moved restlessly about in bed. Although the urine flowed freely, the bowels were not moved. He became gradually more and more prostrate, and died on the following day. Exactly thirty-two hours after he was found in the comatose state by the hospital-sergeant.

In the 'Lancet' of May 14, 1881, a fatal case of acute atrophy (under the care of Dr. Ralfe) is recorded in which no pain or tenderness of the liver was complained of until shortly before death. The chief symptoms being jaundice, nausea, and drowsiness, ending in coma. The patient was a man aged eighteen. His liver after death weighed only 2 lbs. 2 oz. 'Its substance was friable and rotten,' and on section presented a greenish-yellow hue. The gall-bladder was empty and shrunken. Dr. Ralfe thought the disease was 'excited by an indiscretion in diet.'

Twenty years ago when I published my book on the different forms of jaundice, neither I nor anyone else had the slightest idea that acute atrophy of the liver was a disease that ever attacked either infants or aged people.

Lebert in his whole sixty-three collected cases gave only two under ten years of age. Since then,

however, our knowledge of the clinical history of the disease has greatly extended in consequence of several examples having been met with in infants. It is true that Politzer ('Jahrbuch für Kinderheilkunde,' 1860) had related the case of a new-born babe which was on the fourth day after birth seized with jaundice and black vomit, in which the liver became very small, and which terminated fatally on the fifteenth day of the attack; but until more cases of the same kind were reported by other observers little or no attention was given to Politzer's case. Now, however, I have it in my power to cite many examples of acute atrophy of the liver in young children, and the one which I shall select as the most noteworthy is a typical case which was recorded by Dr. Hilton Fagge in vol. xx. of the Pathological Society's 'Transactions,' p. 212. The case is briefly as follows: A little boy aged two and a half years was brought to the out-patient department on two successive weeks with what appeared to be ordinary jaundice, and had a mixture of taraxacum and nitric acid prescribed for him. The day after his last visit a double dose was twice given to him by mistake, and shortly afterwards, as the father described it, he went 'raving mad.' The child soon became unconscious, and died two days after the onset of the serious symptoms. On *post-mortem* examination, 'the liver was found to be in a well-marked state of

yellow atrophy, the cells being destroyed. There were also some balls of tyrosin in the substance of the organ.' Dr. Fagge adds that there was a rash on this child's skin, and that he had before noticed a similar condition in another somewhat similar case. In both instances he observed that the liver on section presented a 'pellucid nucleated matter,' and that he believes that 'the destruction of the hepatic cells is preceded by the formation of a fibrillated substance diffused through the organ.'

I think it may be safely said that the chief feature in the anatomical condition of the liver in cases of acute atrophy is *dissolution of organic structure*. First, a rapid disintegration of the liver cells. Secondly, an almost equally rapid disintegration of their surrounding connective tissue. And thirdly, a disintegration of the coats of the ducts and blood-vessels themselves. At the same time that the liver is atrophied the spleen is in general tumefied.

The name 'yellow' atrophy was given to this acute form of liver disease, from the tissues of the organ after death being stained by the bile pigment of a distinct lemon or orange yellow hue. Quite a different tint from that met with in cases of jaundice from obstruction, when the liver is in general of a greenish-black colour. In acute atrophy, from the tinging of the liver being due to precisely the same pathological cause as the tinging of the blood serum

and skin, the hepatic tissue is not deeper coloured than the rete mucosum.

Having thus fully described the nature of the acute atrophy of the liver which occurs sporadically in temperate zones, I shall now proceed to fulfil my promise of pointing out more fully how closely it resembles the contagious jaundice of the tropics both as regards its etiology and pathology.

Indeed the only point of difference existing between contagious jaundice of the tropics and acute atrophy of the liver of temperate zones is that, while the former almost invariably assumes an epidemic, the latter almost equally invariably occurs only in an isolated and sudden sporadic form. This distinction between these two differently named diseases is, however, one of no pathological importance whatever. For it may be thought and said to be merely due to the climatic differences existing in the localities of their occurrence. Which seems indeed to be proved by the facts :—

1st. That even the most virulent forms of contagious jaundice not only rapidly die out, but entirely cease to be contagious or infectious, when imported, as they occasionally are in ships, into temperate zones.

2nd. Occasionally, even sporadic cases of contagious jaundice occur within the climatic area of the epidemic form of the disease.

3rd. Acute atrophy of the liver sometimes assumes a contagious form even in this country. For, as Graves in his clinical lectures relates, two members of the same family were attacked by the disease about the same time. And the reason why it did not become epidemic was no doubt simply due to the absence of those climatic influences which favour its spread.

4th. Both diseases are attended with a marked hæmorrhagic diathesis. Blood being extravasated into the stomach, giving rise to black vomit, and into the bowels, giving rise to black tarry-looking evacuations.

5th. Both are attended with febrile symptoms and cerebral disturbance.

6th. Both are essentially blood diseases, in the true sense of the word. That is to say, both are the result of an organised disease-germ fructifying in the system. No matter what its mode of introduction into the organism may have been. By direct contagion or otherwise.

7th. Bacilli (by observers, before the different forms of disease-germs were properly differentiated, spoken of as Bacteria: see my papers in the 'Lancet' of June and July, 1881) have been abundantly found, both in the tissues of the liver and in the blood, in all the fatal cases in which they have been looked for.

8th. Neither in the case of acute atrophy nor in

that of tropical contagious jaundice is the liver, though the most prominently, the only organ at fault. Its softened condition being merely one of the local manifestations of the disease-germs' action. Exactly in the same way as the sore throat in scarlet fever, the pustular eruption in small-pox, and the enlargement of the spleen (ague cake) in paludal intermittent fever, are all merely portions of the general diseased action.

9th. The *post-mortem* appearances of the liver are in both identical.

Although jaundice the result of acute atrophy of the liver might in all cases be thought to be a typical example of jaundice arising from a complete suppression of the biliary function—the diminution in secreting substance naturally inducing a diminution in secreting power—it is not so, because, although less bile than usual is secreted, there is nevertheless sometimes nothing like an entire suppression of the biliary function. Which circumstance most probably arises from the fact that, although by far the greater part of the biliary secreting cells are disorganised, there still remains a sufficiency of them to carry on to some extent the secretion of bile. Dr. Hilton Fagge has, I think very judiciously, suggested that, as the whole of the liver is not attacked uniformly, some parts being much sooner affected with the disease than others, the secretion of bile may go on in

the parts less affected, while it is completely stopped in the others, and hence the stools may in some instances actually contain bile. ('Guy's Hospital Reports,' p. 159, 1875.)

The presence, too, of violent cerebral symptoms of bile-poisoning leads to the same conclusion. The cerebral symptoms which supervene in bad cases of jaundice are all in general said to be due to what is called Bilæmia. Namely, to the toxic effects produced upon the nervous system by taurocholic and glycocholic acids, or rather, I should say, their compounds, circulating in the blood, and giving rise to convulsions, delirium, and coma. As this, I believe, is by no means the case in the class of cases now under consideration, and the whole subject of cerebral disturbance in cases of liver disease requires revision, I shall here introduce a separate chapter upon the matter.

The Etiology of Cerebral Derangements in Febrile Forms of Hepatic Disease.

At one time it puzzled me, as it must have done everyone else who has cast a thought on the subject, why—if the theory of bile-poisoning being the cause of the cerebral symptoms in cases of jaundice be correct—it so frequently happens that while cerebral and other nerve symptoms sometimes supervene in a few days, or even hours, after the commence-

ment of the attack, in certain other cases of jaundice from obstruction, where not only the blood, but every tissue in the body—judging from the prolonged duration of the attack and the depth of the discoloration of the skin and urine—must have been saturated with the constituents of the bile for many weeks, not a vestige of nerve derangement is perceptible. Beyond the mere symptoms of prostration and cerebral exhaustion, which are common to all cases of disease associated with malnutrition of the nervous system. A knowledge of these facts drove me to search for some other assignable cause of the presence of head symptoms in certain cases of acute jaundice, and it was a long time before I could satisfactorily to myself account for them. Now, however, I think that I have obtained a scientific and logical solution to the problem. Which is this. In all cases of jaundice where cerebral symptoms rapidly supervene, the *fons et origo* of the morbid state inducing it may be said to be germs. Thus the cerebral symptoms supervene very rapidly in acute atrophy of the liver, in contagious jaundice (yellow fever), and tolerably speedily in severe cases of malarial and paludal jaundice. All of which, as I have already I think conclusively shown, are due to pathogenic germs. Now, if this part of my proposition be granted, the subsequent details connected with it are simple enough of comprehension. Even although, at first sight, from their

very novelty, they may appear improbable. They are as follows :—

All physiologists are agreed that cerebral symptoms and spinal nerve disorders, drowsiness, delirium, coma, convulsions, and paralysis of a particularly well-marked character, follow the artificial introduction into the healthy animal body of both physiological and pathological forms of toxic germs. The only point of difference among them really is as to the proximate cause of the nerve disorder. Some think it due to the non-elimination of excrementitious substances from the blood, and consequent poisoning of the nerve tissue with biliary matters, urea, uric acid, and other such like effete products. While again, the most recently broached theory is that of M. Pasteur, who, from observing the drowsy condition of the fowls into whom he had injected chicken-cholera germs, has been led to believe that, during the life of the parasitical germs in the birds' bodies, a species of narcotic is formed in their blood which produces the somnolent symptoms ; yet be it observed, he at the same time attributes their death to a combination of other causes. To wit, pericarditis, serous extravasations, and asphyxia ('Chemical News,' January 7, 1881). Mark the last assigned cause. For what I am about to say has an important bearing on asphyxiation.

With Pasteur's narcotic theory I have no sym-

pathy whatever. For after having given considerable attention to the spontaneous production of toxic substances in dead animals—from my having in the year 1857 thought I had discovered that hydrocyanic acid spontaneously formed in dog's intestines after death—I fancy I can give a much more philosophic theory of the drowsiness his fowls presented, as well as of the exactly analogous comatose symptoms manifested by my dog killed by snake-poison,[1] and the cerebral symptoms met with in human germ disease, than to imagine that germs manufacture a narcotic in the circulation. I shall relate a personal reminiscence which tends to throw considerable light upon the subject. Everything is said to be fair in love and war, and I think my readers will therefore not feel shocked if I add to my list of arguments in favour of the germ fermentation theory an account of a practical joke which was played upon me during the time I studied in Germany. Everyone knows that when a sufficiency of alcohol in any form whatever enters the stomach it affects the brain. At first it stimulates, and makes lively. Next it acts like a hypnotic, and makes drowsy. While, finally, it narcotises, and makes insensible. The alcohol contained in wine is, or at least ought to be, the direct product of the fermentation of the sugar of the ripe grape. During the early stages of vinous fermentation the

[1] *Royal Society's Transactions*, 1864.

fermenting liquid—'must,' as it is called—is crowded with actively developing living organisms. The unwary drink this sour-sweet fermenting liquid, loaded with life, without the remotest suspicion of its possessing inebriating properties. Without dreaming that while they might drink a whole bottle of the wine made from it without a feeling of cerebral discomfort, a tumbler of this fermenting 'must' may prove a disagreeable narcotic. This I learned by a personal experience made in the following wise.

While working in Kölliker's private laboratory at Würzburg in 1853, my companions were among the *Privatdocenten*—all of whom who are yet alive being now distinguished professors at different German universities—and it was our usual habit to take a long walk into the country after our day's work was done, and before commencing our evening's studies. On one occasion we reached a little hillside village Wirthshaus just as its occupants were busily attending to the fermenting wine; and while we were standing looking at them, one of my companions—now Professor Friedreich of Heidelberg—handed to me a *becher* of must, freshly drawn from the fermenting cask, saying, 'Taste that, Harley, and see how good it is.' It being as agreeable as it was novel to my palate, I drank it all. He then took the goblet, refilled it, and laughingly handed it back to me, saying, 'You have nothing so good as that in

England—take some more.' But, from the smile on his face and his taking none himself, suspecting that there was possibly some trick or another being played upon me, I did little more than taste the second tumblerful; and lucky it was that I did so, as it afterwards turned out. For notwithstanding that there could have been scarcely any alcohol in the must—from the fermenting process having not yet been nearly completed—we had shortly afterwards scarcely begun the descent of the hill, when I began to feel giddy, my ideas to become confused and my gait unsteady. In fact, I was in a state not of alcoholic intoxication, but of 'fermentation inebriation.' Exactly in the condition of the dog into whose blood the puff-adder virus had entered and the fowls into whose circulation M. Pasteur put chicken-cholera germs. The state I call 'fermentation inebriation.' Fortunately, in my case the effects were of short duration; but it was a warning to me never again to give my companions the chance of repeating on me a germ-fermentation experiment.

I totally disagree with Pasteur when he says that the chicken cholera-germs he introduced into the fowls formed a narcotic in the blood, if by the term narcotic he means some alkaloid or another. For such a proposition is not only undemonstrable but, as I shall now proceed to show, wholly unne-

cessary. I believe germs produce the cerebral symptoms he describes exactly in the same way as any gas, fluid, or substance which narcotises by virtue of preventing the brain-substance from being supplied with a sufficiency of properly oxygenated pabulum. In a word, by diminishing tissue oxidation. This is a theory easily understood when it is recollected that if, from any cause whatever, a sufficient amount of oxygen is prevented from obtaining access to the brain, first drowsiness, and then insensibility, is the immediate result. This is most easily demonstrated by making animals breathe harmless nitrogen gas—air simply deprived of its oxygen.

Not alone insensibility, but profound coma and death, soon follow upon an absence of oxygen in the blood. While convulsions, terminating in paralysis, are well known to be the effects of a prolonged diminished supply of oxygen to the spinal cord.

It is to be remembered that marked dyspnœa is a sign in many germ diseases; and not only is it common in miliary fever, scurvy, and purpura, but it likewise existed in a very striking degree in Pasteur's fowls inoculated with chicken-cholera germs, as well as in my dogs poisoned with snake-venom germs.

The explanation of this is, to my mind, very simple, on the ground that the presence of disease-germs in the circulation interferes with the due and

necessary oxidation of the normal nutritive materials of the blood. For example, so far back as in the year 1856, I pointed out in papers entitled the 'Physiological Action of Strychnine' and the 'Direct Action of Strychnine on the Spinal Cord' ('Lancet,' June 7 and 14, and July 12, 1856), the rationale of the convulsive and comatose symptoms arising from toxic agents which destroy the power of the blood-corpuscles to take up the necessary quantity of oxygen for their wants. As I then pointed out, whenever the oxidised materials required as nourishment by the nervous system are either deficient in quantity or impaired in quality, disordered function of the nerves is the immediate result. We have a most striking example of the former condition in cases of hæmorrhage, where an insufficient supply of the oxidised substances is not unfrequently followed by convulsions; while the latter is exemplified in cases where oxygen is prevented from entering the blood, and consequently the organic substances fail to become oxidised and fitted for their peculiar office. Lastly, nerve-disorder occurs when even both the oxygen and the organic substances are present, but where the oxidising process is either partially or totally arrested by the presence of a foreign substance possessing the property of hindering the constituents of the blood from combining with oxygen. Derangement in the functions performed by the mole-

cules of the nervous system occurs just as surely in the latter set of cases as when either the oxygen alone, as in the second, or both the oxygen and the oxidisable materials, as in the first instance, are wanting. Germs act in the third of the ways cited —that is to say, they have no *immediate* effect upon the nervous system themselves, but only act indirectly through the power they possess of using up the oxygen which ought to go to oxidise the constituents of the blood, and thereby fit them for the purposes of nerve nutrition. This mode of action is readily accounted for by the fact that all germs have a very active respiratory function—absorbing oxygen and exhaling carbonic acid exactly as other animals and vegetables do—and in direct proportion to their respiratory activity is their deleterious action on the constituents of the blood, and through it on the nervous system of their host. For the more oxygen the germs consume, the less is there left to enter into combination with, and oxidise the host's tissues, and keep them up to the proper working standard. As the just appreciation of this novel theory requires not only physiological knowledge, but an intimate acquaintance with the life-history and physiologico-chemical actions of disease-germs, and I cannot afford space to go into more details, I beg those of my readers who are not well versed in the matter, before reading further, to peruse the chapters on the Pyrexia

and Fœtors of Disease. For, as I explained in the Preface, as I have no space to waste in unnecessary repetitions, many of my data and arguments must be considered not in fragments, but as one great and indivisible whole. Many poisons, I doubt not, exert their influence on the nervous system in an equally indirect manner; for I have found that hydrocyanic acid, chloroform, nicotine, alcohol, ether, morphine, and several other narcotics, have the power of destroying the property possessed by the organic constituents of the blood of absorbing oxygen and exhaling carbonic acid. And as strychnine, which is a markedly convulsion-producing poison, has this power, as I showed in the papers above referred to, I believe its physiological action on the nervous system is due to its preventing, like germs—though by another process—the nerve-tissue of the whole cerebro-spinal system receiving properly oxidised pabulum for its nourishment, and, as a natural consequence, its functions are thrown into disorder, and delirium and convulsions occur; just as they do when it receives an insufficient supply of blood pabulum—as happens in cases of hæmorrhage.

In cases of fevers, again, there are additional causes of nerve disorder. Among the most prominent of which is increased temperature. No one doubts the power of heat in producing nervous disturbance, since all know the rationale of sunstroke;

and it is consequently easy to understand how the heat developed in the body by the germs' fermentation must act prejudicially upon the nerve-tissues, already weakened from being supplied by pabulum not properly prepared for their wants.

The lassitude, nervous prostration, and want of mental as well as bodily power, which are so characteristic of all germ diseases, ought to be attributed to the above causes. For as soon as the crisis of the disease is passed and the germs are eliminated, all the visible signs of vital exhaustion rapidly disappear; thereby showing that they only depended on the existence of temporary causes such as I have shown in the manner above described would be produced by germs.

Before quitting the question of the etiology of germ-action in producing nerve disorder, I must add a few words on the rationale of the cause of the sudden production of nerve symptoms and rapidly fatal ending in certain cases of diabetes which appear to be so absolutely unaccountable as well as so startling to the uninitiated, from the patients being often apparently in their ordinary state of health one day and yet dead the next, as the consideration of them throws some light on the etiology of the cerebral disturbance met with in certain cases of liver disease. In the 'British Medical Journal' of November 1, 1879, it is stated that Dr. Jules

Cyr, in the December and January numbers of the 'Archives Générales de Médecine' for 1877–8, details thirty-two cases of sudden death in diabetes, collected from various sources. He considers that there are at least five different conditions to which these may be ascribed: 1. The formation of acetone in the blood under conditions nearly unknown—acetonæmia; 2. The accumulation of excessive quantities of sugar in the blood—hyperglycæmia; 3. The retention of urinary solids or water in the blood—uræmia, dropsy of the ventricles; 4. Atrophy of the cardiac muscles; 5. Cerebral anæmia. Of the thirty-two cases, twenty-one are stated to have died comatose; in a few the mode of death is not stated; in others there is no mention of coma; but this large proportion shows the relative frequency of this mode of death. Dr. Balthazar Foster has in the same journal, of January 19, 1878, urged the probability of acetonæmia being the cause of death in a large number of diabetic cases. This theory he supports by quoting three cases from his own practice; in the first no smell of acetone was noticed in the breath, but the blood of the patient, examined after death, was of a peculiar pale colour and creamy consistence; under the microscope, the blood-corpuscles were broken down into a granular material, which he subsequently found could be artificially imitated by treating blood with acetone. In the other two cases

there was a strong odour of acetone in the breath of the patients. Dr. Foster alludes to the objection which has been made that, in many cases, no odour of acetone is perceptible, and replies that a temperature of 100° Fahr. is necessary to volatilise acetone. Kussmaul ('Deutsches Archiv für klinische Medicin,' 14 Bd., 1874) has gone very fully into this question of acetonæmia, and from his experiments concludes that it is not possible to believe in a theory of acute intoxication from acetone, but that chronic poisoning by this substance may so affect the nervous system as to render it liable to take on an acute form, just as chronic alcoholism may suddenly explode in delirium tremens.

As the onset of the dangerous and fatal symptoms in these cases of diabetes always occurs suddenly, and when, as far as one can see, the pathological conditions existing in the patient are not in the least different from what they have been for months or it may even be for years previously, it is clearly evident that *some change* has suddenly been brought about in the patient's body by the introduction of an entirely new element. That new element is, to my way of thinking, the accidental and sudden introduction into the system of ferment-germs.

The blood of the diabetic patient is full of sugar. The temperature of his body is exactly the one of all others the most favourable for the fermentation

process ; so that nothing further is wanting to induce a fatal fermentation of the blood than the presence in it of ferment-germs. They at length, I believe, from some cause or other, find accidental admission, and instantly, as a natural consequence, fermentation begins ; and in a few hours, or it may even be minutes, the blood's constituents have undergone a sufficient amount of morphological and chemical transformation to induce all the usually described chain of nervous symptoms which culminate in the sudden death of the victim.

Why, it may be asked, if this theory be correct, did the ferment-germs which entered my stomach along with the must I drank, and which must have got into my blood, or otherwise they could not have produced the chain of effects which I have described under the title of 'fermentation inebriation,' pass so soon away, and not only not kill, but even leave in me no bad after-results ?

This question, I think, is very easily philosophically answered. Had I been a diabetic patient, and my blood loaded with sugar, the must-germs would in all probability, having found suitable pabulum, have increased and multiplied, and thereby set up an amount of fermentation changes in my blood sufficient in all probability to have killed me ; but I was not a diabetic patient, and my blood was not loaded with sugar. Consequently, as the germs

found but little food for their growth and multiplication within my circulation, they could neither set up within it a rapid and, to me, fatal fermentation, nor could they themselves live there. From there being no suitable pabulum for them to live upon, they were simply starved to death ; and, as a consequence, the slight disturbance they were at first able to create soon entirely ceased, and their involuntary host speedily felt no further effects from their temporary sojourn in his system.

I am not quite sure if delirium tremens—the result of alcoholism—may not be a condition of system due to the action of germs. Germs, as we know, are essential to the production of all fermented intoxicating liquids, and the peculiar odour of the perspiration in delirium tremens points to germs having something to do with the symptoms. It is usually described as being of a saccharo-alcoholic description. Not unlike that arising from fermenting must.

Again, I attribute the local gangrenes and sloughings, which occasionally take place in typhoid cases and diabetic patients, to the germs producing them being of the species that thrives locally. Some germs, we know, have a tendency to produce only local effects, and others, again, general. In the way typhus-fever germs always produce a general disease, while vaccine germs give rise usually to a merely local affection.

The kind of germ in the two sets of cases determining the nature of the result.

The delirium which is such a prominent concomitant of contagious jaundice, and atrophy of the liver, and other forms of acute and febrile hepatic disease, is, I believe, as in diabetes, due to the presence of fermentation-germs in the patient's blood. Whereas the cerebral symptoms which accompany the chronic forms of jaundice from obstruction and suppressed secretion are due to the condition called bilæmia, as above interpreted.

Treatment in Cerebral Cases.

It is well to remember that in cases of hepatic disease, with delirium and other forms of head symptoms, associated with marked pyrexia, there are other ways and means of reducing the circulation, the temperature, and the head symptoms, than by the administration of quinine and other forms of germicides spoken of in the general chapter upon remedies—to wit, the administration of aconite or digitalis. Or, if these are considered inadmissible, the direct application of cold to the head in the shape of an ice-bag or cold-water head-irrigator. In most cases, James's Powder, as well as the liquor ammoniæ acetatis, is useful in subduing violent delirium.

When suddenly called to the bedside of a delirious

patient labouring under jaundice, the pathology of which is obscure, it is always well to bear in mind before prescribing that dark stools are not, *per se*, proof of the presence of bile in them ; for, as already shown, the black bilious-looking colour of the motions may be due, not alone to the presence of blood in them, as happens both in cases of contagious jaundice and acute atrophy, but to the medicines the patient has been taking. That delirium in the case of contagious jaundice is accompanied with intense febrile symptoms, and in the case of acute atrophy by greatly diminished dull hepatic area. When, again, the delirium is the result of pyæmic jaundice, it differs from that occurring in both of the preceding cases, not alone in having been slow and gradual in its advent, but in having been usually preceded by distinct symptoms of hepatic inflammation. Be the cause of the delirium, however, what it may, it invariably indicates the existence of danger to life, from the circumstance that it is due to blood-poisoning. In the case of acute atrophy and pyæmia from a form of albuminoid putrescent fermentation. In contagious jaundice, and in malignant jungle-fevers, from a malarial or paludal vegetable germ-growth. Consequently, in all cases of delirium occurring in the course of hepatic disease, it is good policy to favour, in every possible way, the elimination of the poisoning materials from the blood, and this

is best done by increasing the action of the skin as well as of the kidneys. Brisk cutaneous friction, a hot-air, a steam, or a hot-water bath, ought therefore to be had recourse to, according to the constitution and strength of the patient, at the same time that diuretics, such as squills, digitalis, broom-tops, or sweet spirits of nitre, are administered by the mouth.

In some cases the stools are loose, but not always, and in the latter case their free action should always be encouraged by the administration of vegetable purgatives, such as castor-oil, colocynth, or rhubarb, when mercurials are contra-indicated.

As the lungs greatly assist the skin and kidneys in eliminating biliary products, and thus mitigate cerebral symptoms, the free access of dry fresh air is an adjunct of great therapeutical value in all cases where there are signs of blood-poisoning.

As I have thus far digressed from the immediate consideration of atrophy of the liver, I must not let the opportunity slip of saying something on the pyrexia of hepatic affections. For I am of opinion that it is quite as great a clinical pathological puzzle as the rationale of cerebral disturbance, if not even a still greater. From the causes of pyrexia as a whole not only being but imperfectly interpreted, but actually, I believe, entirely misunderstood. I shall therefore here bestow another entire chapter on its consideration. And shall treat the question on the

same broad basis as I have done cerebral derangements, so as to make my remarks applicable to all kinds of pyrexia in general, fevers, &c.

The Etiology of Pyrexia.

The etiology of the abnormal temperature met with in a variety of different diseased states of the human system is a subject which has puzzled the philosophic physician ever since clinical thermometrical studies began. From the ever recurring question being 'Why does the temperature of the human body fluctuate so greatly in disease without any apparent assignable cause, when it remains so stationary during health in spite of many well-marked assignable reasons?' Scientists as well as able philosophic physicians have at various times and in different ways tried to explain the reason why the temperature of the healthy human body is at the North Pole, with an external and breathing temperature of 46° Fahr. below the freezing point, exactly the same, to within a few tenths of a degree, as it is at the tropical equator with an external temperature of 136°. Giving thus a range of difference of 150 degrees. This to the reflecting mind is an astounding fact. When it is remembered that the temperature of the diseased human body often varies as much as 10° in the space of a few hours, not alone while the external tempera-

ture remains stationary, but even when it is reduced to more than a half of the temperature of the body itself.

In perfect health the temperature of the human body is subject to periodic fluctuations, and these physiological diurnal fluctuations of temperature were ascertained by Dr. William Ogle (St. George's Hospital Report, 1866) to be at their minimum at 6 A.M., and maximum between 2 and 4 P.M. The difference ranging in different persons from a half to a whole degree.

These physiological and pathological facts, even when regarded by themselves, convincingly show that the biological laws regulating the temperature of the human body in health are entirely set at naught in disease. So that we cannot apply to them for a satisfactory answer to the question of 'What is the cause of abnormally high temperatures ?' but seek for its explanation not alone outside the pale of healthy action, but probably even beyond the confines of the human body itself.

Indeed, I think I may as well here at once further state that it is my opinion that the reason why the causes of the high temperatures in disease have not as yet been satisfactorily explained arises in great measure from two circumstances.

1st. That all abnormal high temperatures have been placed in the same category, and consequently

been attempted to be explained by one and the same theory.

2nd. From cases of pyrexia having been supposed to originate in, and be due to, abnormal nerve action.

Pyrexia, for example, as defined by Aitken in his able work on the 'Science and Practice of Medicine,' is: 'A complex morbid state which accompanies many diseases as part of their phemomena more or less constantly and regularly, but variously modified by the specific nature of the diseases which it accompanies. It *essentially consists* in *elevation* of *temperature*, which *must arise* from an *increased tissue change*, and have its *immediate cause* in alterations of the *nervous system*.' (The italics are mine.) This definition is founded upon the published views of Parkes, an English physician; Virchow, a German pathologist; and Bernard, a French physiologist whose opinions upon the etiology of pyrexia are those which within recent years have been received with most general favour all over the world. These writers having attributed the increase of bodily temperature in all cases of disease, even in fevers—that is to say germ diseases—to increased blood and tissue metamorphosis. *Induced by an abnormal action of the nervous system.* Founding this opinion on the well-known fact that section of the eighth pair of cerebral nerves in the neck of a rabbit not only produces an increased flow of blood to that side of the face and head, but an

increase of temperature. This nerve theory being further supported by the fact, that so soon as the nerve influence is artificially restored by galvanising the upper end of the divided nerve, the turgescence of the vessels diminishes, and the temperature of the parts supplied by them falls.

Now I may add that, on these experimental physiological facts, the generally accepted nerve inducing blood and tissue metamorphosis theory of high temperature in certain (though not in all) forms of disease may be scientifically accounted for. On the supposition that, on account of the centripetal nerve lesion, the inhibitory action of the nervi vasorum is lost. In consequence thereof, the vessels lose their tonicity, and allow the blood to rush through them in increased volume, and probably at the same time also with increased speed, from the heart's action being likewise accelerated.

Assuming then that this theory is physiologically and pathologically correct, it gives a satisfactory explanation to all the cases of elevation of temperature arising from nerve irritation and nerve lesion, such as those reported by Dr. Goodridge, where, in a case of softening of the pons Varolii, the thermometer indicated a temperature of 103°. In Dr. Little's case of cerebro-spinal meningitis, where it stood at 106°, and in Mr. Teale's case of concussion of the brain with spinal injury where it rose to 108° Fahr. As is seen, I am not quoting the extremely high readings

of the thermometer which occurred in those cases, being in Mr. Teale's 122°, and in Dr. Little's 133°,[1] as they are now regarded by many as fictitious, and it is not with the etiology of fictitious but of real high temperatures I am now dealing. So I leave the exceptionally high thermometrical readings of the above-cited cases aside, and merely cite such as may be accepted by us as real.

Now I come to an important point—which is, that, while I believe the nerve inhibitory theory yields a satisfactory solution to the probable cause of the bodily temperature being increased in all cases of idiopathic as well as traumatic nerve derangements, I consider that it is not only totally inadequate, but totally inapplicable, as a rational explanation of the cause of the increase of bodily temperature occurring in any of the other kinds of diseases in which it is usually met with—to wit, those grouped together under the name of true pyrexial or febrile diseases. Which all belong to the germ class of infectious, contagious, and inoculable affections.

I will even go so far as to say that the nerve theory of high temperatures when applied to any single one of them is radically wrong, and even, at the risk of startling some of my readers, add that the increase of bodily temperature in such cases has

[1] See *Medical Times and Gazette*, November 5, 1881.

possibly—ay, even probably—primarily nothing to do with the patient's nervous system whatever. Nay, even more, I am actually prepared to assert, as well as to prove, that the heat of the body in pyrexial diseases of the infectious, contagious, and inoculable class, has primarily nothing whatever to do with the patient's own body, but entirely originates in, and is dependent upon, the activity in the development of parasitic germ life.

This may appear to be an astounding proposition, but I venture to say it is only astounding because it is new; certainly not because it is untrue, as the sequel will show.

In my mind, the nerve and tissue metamorphosis theory of high temperature received its death-blow when, on proceeding to make the necropsy of a well-developed plump young woman of 20 years of age, who died on the night of her admission into University College Hospital of acute pericarditis supervening in scarlet fever with cerebral symptoms, I found the tissues of the body quite warm. Notwithstanding that the body had lain for several hours in the cold post-mortem room. I was told that she had died during the night, and, as is well known, the term 'during the night,' in hospital parlance, is in general intended to mean between the hours of 11 P.M. and 4 A.M. So that this woman had probably been dead not less than ten, and not more

than fifteen hours, as I made the *post-mortem* at the usual hour of two o'clock.

Well knowing that the human body cools rapidly after death—sometimes, indeed, the extremities and exposed parts becoming stone cold even before the vital spark has fled—I was surprised to find this woman's body quite warm, that the chest steamed when opened like that of a newly-killed ox, and that the heart felt hot to the touch. From my not then perceiving the great clinical importance of finding that a dead human body in certain forms of disease might retain an abnormally high temperature, and having no clinical thermometer at hand, I neglected to send for one. Yet I think from my familiarity with manual temperature-taking, that in this case it was certainly not less than 100°—probably even as much as 102°—notwithstanding that she had lain several hours dead in a cold room. Which fact is of course of immense importance when taken into consideration with the high temperature theory of the present day. For seeing that life was extinct, and consequently the circulation not only completely arrested, but all nerve force totally annulled, how could it be possible that the temperature of this woman's body could be maintained on the nerve influence and tissue metamorphosis theory? Simply impossible. And many and many a time since my eyes were opened to the importance of this fact,

have I reflected on this, to me, extraordinary observation, and long and patiently have I waited in the hope of meeting with a similar case which I might be able to thoroughly investigate.

Fortunately at last my wishes have been fulfilled. Not, however, by my meeting with a similar case, but by what is equally good, if not even better—finding one ready to hand by Professor Wunderlich. Which has not only the advantage of being reported by a trustworthy and perfectly independent observer, but likewise that of not having attracted his attention to it in the important way it has mine.

Professor Wunderlich gives to his case the title of 'rheumatic tetanus,' and he says that after the patient's death the temperature did not alone not begin to diminish, but actually went on increasing until within an hour after death, when it was found to have risen from 112·5° to 113·8°. That is to say, 1·3° Fahr.; and, further, the temperature of this corpse did not fall even so much as to the normal standard until thirteen and a half hours after death.

Here then is proof positive that nerve influence is not the sole cause of the temperature of the body being increased in all forms of disease, as stated by Bernard, Parkes, Ludwig, Virchow, and a host of subsequent writers. For in one of the above-cited cases we see that not alone was the high temperature of the body maintained, but actually increased

after death. When not only the circulation of the blood had completely ceased, but all nerve influence had been likewise totally extinguished.

The factor of death thus gives the *coup de grâce* to the commonly accepted theory of the probable cause of abnormally high temperatures in certain forms of disease being due to a hyper blood and tissue metamorphosis, induced through the intermediary action of a living nervous system.

Having thus demolished the nerve theory of pyrexia in cases of germ disease, and shown that it is only applicable to the local and general increase of bodily temperatures arising in a limited number of cases as the direct result of nerve irritation or lesion, I shall now attempt to establish another theory of abnormal temperature, on a soundly scientific and demonstrable basis, and one, too, which shall be applicable to all the forms of pyrexia met with in infectious, contagious, and inoculable diseases. In order to save time, I shall begin by at once stating that I believe that in as far as the etiology of the increase of bodily temperature in germ diseases is concerned, the nerves, blood, and tissues of the human body merely play the part of passive agents. The abnormal heat of the body being produced by, and totally depending upon, the development, growth, and multiplication of the germs engaged in producing the disease. The pyrexia being in

fact the outcome of the germ's life itself, and the rise in the temperature of their host's body nothing else than the chemico-physical effects of the heat developed by the germ's respiratory activity. Animal heat being, as we know, one of the products of tissue oxidation.

These remarks may probably appear to some of my readers as absurd; but like many other statements the apparent absurdity only exists in their novelty, as I shall now proceed to show. But in order satisfactorily and conclusively to prove the justness of the ideas I am endeavouring to promulgate I must request my readers to allow me to address them for a few minutes not as physicians and surgeons, but purely as physiologists. For in the present, as in many other cases, my pathological theory is entirely dependent for its logical explanation on the twin sister science of physiology. A thing not to be wondered at, seeing that both pathology and physiology are the offspring of one common parent—biological science. I will not, therefore, offer any further apology for putting practical medicine entirely aside for a few minutes, and turning the train of thought into a purely physiological chemical groove in which I expect to meet with a talisman by whose aid I may perhaps be enabled to unlock the door to the etiology of the high temperatures met with in cases of infectious, contagious, and inoculable diseases.

Of course I may take it for granted that my readers are all conversant with the comparatively speaking new doctrine that fermentation, whether occurring without or within the animal body, is simply the direct effect of living organisms, generically named germs, on organic matter; and that the fermentative action is not in any way dependent upon the chemical constitution of the germs themselves, but upon their attribute of life, and life alone. And further, that as disease is simply the outcome of misplaced healthy action, in a precisely similar manner it is the life and the life-manifestations of disease-germs which alone possess the power of producing what are named pyrexial diseases. These points being admitted, I have further to call attention to the fact that when I speak, as I shall presently have to do, of the respiratory function of germs, I mean respiration in its broad and philosophic sense. Not the merely limited physical process of the inhalation and exhalation of gases by special organs, but the chemical combination of oxygen and the exhalation of carbonic acid gas by the solid constituents of all animal fluids, as well as of all animal tissues. Which function is the inherent attribute and inseparable concomitant of all active animated existence. Be it remembered that what we call active, stands in contradistinction to what we name passive life in a direct and progressive ratio to the chemico-physio-

logical activity of the indispensable function of respiration. From the fact that 'life,' animal or vegetable, in a strictly philosophic sense is merely the collective visible functional manifestations of organised tissue and fluid oxidations. The more rapidly tissues and fluids are oxidised, the more active are the functional manifestations which we collectively denominate 'life,' and as a certain proportion of heat is set free from every molecule of material oxidised—whether it be in the muscle, in the bone, in the brain, or in the blood of higher, or in the homogeneous tissues of lower animal and vegetable species—it follows as a natural corollary that the quicker the development and multiplication of germs, just as of other animals and plants, the greater must of necessity be the absolute oxidation, and consequent amount of heat evolved by them.

Thus it is that the parasitic germs themselves, by virtue of their own vital activity, develop, among other things, heat, at the expense of their host's component parts, and, as a natural sequence, raise the temperature of its body. No matter whether it be, as in the case we are now considering, a living human body, or a dead inanimate object. For precisely the same thing occurs when the germ's host happens to be a milk-can or a soup-tureen.

This apparently strange assertion is as comprehensible, when scientifically viewed, as is the heating

of a pot of water by a coal fire; and I shall now endeavour to substantiate it, as far as it is possible to do so, by a legitimate process of reasoning from demonstrable analogy. Which, although it may be regarded as a roundabout way of proving a fact, is by no means an unsatisfactory manner of establishing a scientific principle. For be it remembered that presumptive is invariably of equal value to direct scientific evidence, when it rests upon a demonstrable basis.

As it is generally admitted that in establishing the validity of an argument one single incontrovertible demonstrable fact is of more intrinsic value than tens of thousands of assailable statements, I shall, for the sake of brevity, content myself with adducing one crucial illustration of the correctness of my views.

Taking for granted that it is now admitted that germ fermentation is the chief factor in the production of pyrexial diseases, I may at once proceed to say that few persons have the faintest idea of the enormous amount of heat developed by germs during their active fermentative life, and therefore I expect that it will not a little astonish some of my readers to be informed that the amount of heat developed and evolved by germs during the fermentation of certain organic substances amounts to no less than 65° Fahr.! This I can prove by referring to the

results of the experiments recorded by a professional chemist.

Professor Atkinson in his paper—which, fortunately for my argument, has nothing whatever to do with medicine—on the fermentation of rice in the formation of the Japanese diastase, 'kôji,' by the spores of the *Eurotium oryzeæ*,[1] tells us that when the temperature of the external air was 41·5°, that of the fermenting mass itself was 106·6° Fahr. ; and that the heat set free and evolved from it during the process of fermentation was actually sufficient to raise the temperature of the chamber in which the operation was conducted to double that of the external air—namely, from 41·5° to 83° Fahr.

If such then occurs in a cold dead fermenting vat, how can we be surprised that an analogous kind of fermentation taking place in a living human body should suffice to raise its temperature a few paltry degrees beyond the normal range, when it is remembered that the human living body, so long as it contains the requisite materials for the germs to live and develop upon, plays nothing more than precisely the same part as a dead containing-vessel, such as a vat or anything else, does ? The tempera-

[1] Atkinson says that the change taking place in the rice during the process of the fermentation of kôji—which is a powerful ferment, used by the natives to raise their bread, make soy sauce, and saké liqueur—is the conversion of the insoluble albuminoids of the rice into soluble. During which oxygen is absorbed, and carbonic acid freely exhaled. (*Proc. Roy. Soc.* No. 213, p. 299.)

ture of the human body, like that of the vat, being only maintained above the normal standard so long as it contains germ-pabulum. This is all the more readily understood when viewed in connection with what I said regarding the immensely rapid development of putrefactive germs in the human body after death, at page 598 of the 'Medical Times and Gazette,' November 19, 1881, where I related three cases of rapid putrefaction, in one of which the development of the germs and the activity of their respiration was actually made visible to the eye by the evolution of gas. Which of course, as germs breathe, is an inseparable concomitant of their active existence.

There is yet another important point in connection with the fermentation of kôji, which is of great interest to us as practical physicians, as it may probably furnish us—though it is not yet explicable—with a clue to the cause of the chilliness, often amounting to rigors, experienced by patients at the onset of such germ diseases as malarial jaundice, smallpox, typhoid, typhus, relapsing fever, &c., which has so long baffled all human ingenuity to fathom. For Atkinson says that on the second day after the raw rice has been mixed with the germ-spores, and *before active life* in the kôji has actually begun, its temperature, from some unknown cause or other, is suddenly reduced half a degree lower than that of the surrounding atmosphere; which, from its having

been said to be 41·5°, gives the kôji one of only 41° Fahr. This is certainly, to say even the least of it, a curious fact, and undoubtedly, to us, exceedingly interesting from a clinical point of view ; for be its ultimate explanation what it may, it is the undoubted typical analogue of the chill patients experience at the onset of germ diseases.

I have yet to call attention to another equally remarkable circumstance in connection with the fermentation of kôji—namely, that just as the temperature of the human body labouring under germ diseases varies at different times of the day, in precisely like manner does the temperature in a vat of kôji vary during the fermentation process. This fact can surprise no one who is conversant with natural periodic biological law. For he must at the same time know that while no effect in animal or vegetable nature, no matter how trivial it may be, ever occurs without a cause, every similarly chemically and morphologically constituted organic and organised, simple or compound, substance or organ—vegetable or animal, whether living or dead—when placed under the same conditions, invariably displays the same properties. So that there is nothing surprising in the fact that the same periodic changes should equally take place in a living human body and in a dead wooden vat—changes denominated vital in the one case, and chemico-physical in the other—

when the one instead of the other happens, by a mere alteration of circumstances, for the time being to be the germ's host.

Moreover, I explain the diurnal variations which I am about to show take place in the fermenting vat according to the great and universal law of periodicity of function in every animated thing, be it a plant or an animal—a law very little attended to, and still less understood. For the microscopic objects which we denominate germs, and which are now regarded by the advanced school of pathologists as the immediate cause of all pyrexial diseases, minute though they be, nevertheless, I believe, possess the same physical and chemical attributes in proportion to their size and constitution as man himself, who is thought to stand at the pinnacle of animated nature. And just as every function of the human body is essentially more or less regularly periodic, so, in like manner, is every germ-function. Hence the similarity in the fluctuations of temperature in diseased man and in the fermenting-vat, when he or it happens to be the germ's host. As I deem this fact to be one of very great importance in studying the etiology of human abnormal high temperatures, I subjoin a table of the diurnal fluctuations in the temperature of fermenting kôji, which I have drawn out from the observations recorded in Professor Atkinson's purely chemical paper.

Date	Hour	Outside temperature	Temperature of Chamber	Temperature of Kôji
		Degrees Fahr.	Degrees Fahr.	Degrees Fahr.
Dec. 5	8 A.M.	40·7	—	104·8
	2 P.M.	49 5	83·0	91·8
„ 6	8 A.M.	41·5	83·0	106·6
	1 P.M.	50·0	82·5	104·1
„ 7	9 A.M.	38·5	82·5	104·2
	2 P.M.	51·0	82·0	93·6
„ 8	8 A.M.	37·5	82·5	100·0

As is seen, the last column reads exactly like the temperature table of a case of pyrexial human disease. So like, indeed, is it to one that were it placed in a clinical report, without any notice being made of its origin, the reader would almost for a certainty imagine it was a record of the human bodily temperature fluctuations.

These remarks will be all the better appreciated if the reader will kindly refer to what I said in Chapter II. on the Periodicity of Disease in the 'Medical Times and Gazette' of November 1881. Where I called attention to the interesting fact that not only is every function of animated existence more or less distinctly periodic, but that every movement in nature likewise obeys one great and fundamental periodic law. Even the migrations of the ova of the *Filaria sanguinis hominis* show the same periodic law, for they are found to abound in the patient's blood between the hours of 9 P.M. and 6 A.M., and are totally absent from it between 9 A.M. and 7 P.M. And the temperature of the body is in

cases of filarial migrations found to be highest at the hours when they are most numerous in the circulation.

Although it has been long known that various species of filaria produce a variety of diseases, it was only last year that we became aware that a small microscopic species could induce jaundice, by the discovery of Dr. Evans that the disease among horses called 'surra,' which prevails in the Derajat, west of the Indus, is due to filaria. Dr. Evans describes the disease in the following terms : 'A specific, parasitical, non-inflammatory, enzootic blood-disease, characterised by fever with jaundice ; petechiæ of mucous membranes, especially of the eye and vagina ; dropsy ; albumen sometimes in the urine ; great prostration of strength, rapid wasting, and with a specific parasite in the blood during life ; but no characteristic structural organic lesions are found after death. It may be transmitted by the subcutaneous injection of blood, and by drinking freely blood containing the parasites alive, but is not contagious or infectious in the ordinary way. The average duration of the disease is probably not less than two months, but reliable statistics are wanting upon that point.' The parasite is described as having a round body tapering in front to a sort of a head, and ending behind in a tail, about three or four times the diameter of a white

corpuscle in length, and one-eighth to one-tenth in breadth. He asks whether the disease has been known to exist in any other part of India; and, if so, whether the localities were marshy and the water unwholesome, and whether the animals had been particularly exposed to the sun or to fatigue before they showed symptoms of the disease.

On reading the above description of the filaria, I was forcibly reminded of a specimen of jaundice urine from an old Indian officer I once examined, in which were found a few exceedingly minute worms, which I exhibited to my histology class at University College under the title of *Filaria infinitesima*. First, because, though of minute size, they still exactly resembled in shape and form an ordinary round filarial nematoid worm—such, for example, as the strongylus, represented in fig. 4, which is taken from my 'Histological Demonstrations'[1]—and secondly, though a real worm, from its being in many cases not bigger than an ordinary human spermatozoon. The form and appearance of which it so closely resembles that if two headless spermatozoa were united together by their necks, and their tapering tails left free, they would conjointly be a good portrait of a *Filaria infinitesima*, which they resemble equally well in pellucidity and shape.

In case some of my readers may be led from the

[1] Longman & Co., 2nd edition, p. 247.

above description to imagine that these worms may be a form of spirilli, I may as well mention that I have never seen them put on a corkscrew-like appearance, never go beyond forming the single anterior and posterior undulating S-shaped curve of the true nematoid worm. Whereas fig. 10 shows that spirilli are characterised by many twistings.

On examining these small worms, I could not help feeling that, morphologically speaking, they stand at one end of a type of parasites, while spirilli stand

FIG. 10.

Spirilli.

FIG. 11.

Sperm-cell containing mature spermatozoa, from common house-sparrow.

at the other, and that the intermediate species of animated creation might be said to be a spermatozoon. As in the spermatozoon of the common house-sparrow we possess a united type of both. For while at its one end it presents the form of the spirilli disease-germ, at the other it is a true representative of the *Filaria infinitesima*, as may be seen in the annexed woodcut (fig. 11), taken from my 'Histological Demonstrations,' showing a parent sperm-cell full of spermatozoa.

There is yet another point of analogy in the clinical history of febrile diseases the result of parasites and ordinary fermentation, which I desire to call special attention to, and that is, the definite periods of their course. Every pyrexial germ-disease which does not end fatally, if left to itself, runs a definite course, and terminates, of its own accord, in a certain number of hours or days. In precisely like manner does every known species of fermentation, whether it be that of dough, sauce, soup, beer, or wine—and I believe from a precisely similar cause: namely, the life's career of the germs being in all cases definite. And just as external physical circumstances, as well as internal constitutional causes, hasten or retard the progress of pyrexial disease, so in like manner do they hasten or retard the various forms of fermentation. This opinion is not a mere hypothesis. It is a legitimate scientific theory, for it is demonstrable, and it requires neither any great stretch of imagination to understand that when normal ferment-germs have ceased to live, they cease to be able to produce fermentation—in a lump of dough, in a barrel of beer, or in a vat of wine—nor any violent effort to comprehend that with their life's career fermentation ceases in the human body, and when fermentation ceases the disease it gave rise to is at an end.

Finally, now comes the question: Does the germ

theory of pyrexia explain what the nerve theory fails to do: namely, the cause of the high bodily temperature being in certain cases maintained after death? Most decidedly it does: and how, I shall now proceed to show.

The mere fact of the pyrexial temperature of the tissues of the human body being maintained for many hours after the death of the patient shows that the heat-producing agents act independently of the life of the patient. For otherwise it would be impossible for their thermometric effects to continue so long in operation after all the human vital actions had ceased. The fact is easily accounted for in the following wise.

As will have been noticed, according to the views I take of the causes of high temperatures in germ diseases, the germs, and the germs only, are the real heat-producing agents. The heat they evolve and communicate to their host's body being the direct product of their vital activity, and quite independent of the life, death, or even the nature of their host. So long as the host contains the pabulum necessary for their growth and multiplication, it matters not one whit to them whether it be a living animated being, or a dead inanimate inorganic thing, in which they carry on their operations, from their life's activity not depending upon the nature of their host, but of their host's contents. For as germs are mere parasites, they are perfectly independent

beings, in as far as the nature or functions of their host are concerned, so long as its nature and functions are not incompatible with their existence. Consequently germs can live, grow, multiply, and evolve heat, even better in a dead than in a living human body, for the very simple reason that, while the former is merely the passive receptacle of their pabulum, the latter is in addition an active agent in their destruction and elimination—as I pointed out in the 'Medical Times and Gazette'—and the only reason that I can see for the pyrexial heat of the body not being usually maintained after death is because the pabulum has, in the majority of cases, become exhausted ere the host dies, and with the extinction of the host's, that is to say, the patient's, life, ceases the generation of a fresh supply.

In those exceptional cases again where the abnormal temperature is maintained for several hours after the patient's death, as in Wunderlich's and my case, I imagine that there existed in the patient's system a superabundant supply at the moment of death, and so long as the supply lasted the germs went on growing and multiplying and evolving heat, just as they did while the host was alive, and the amount of heat they evolved was sufficient, like that evolved from the kôji, to maintain the dead host at a high temperature. A similar amount of heat being contributed by the germs to the dead as to the living

tissues of the body, equally independently and in precisely the same manner as the heat was contributed to the chamber in which the fermentation of the kôji was conducted.

Thus then, in conclusion, I think I may venture to say that the fact of the temperature of the human body after death having been maintained in the cases cited, has, by a logical and philosophic process of reasoning from analogy, yielded a probable clue to the abnormally high temperatures met with in diseases of the infectious, contagious, and inoculable class. Having now shown that there are at least two distinct causes at work in the human body producing abnormally high temperature, I may mention that the reason why in all pyrexial diseases the bodily temperature does not reach the same point is readily explained by the fact that all diseases are not the product of the same germs, and different kinds of germs produce different amounts of heat. For example, the fermentation of horse-dung is due to one species of germ, that of the kôji to another, and while both develop a sufficient amount of heat to elevate the temperature of the chamber in which the fermentation is conducted, the amount of heat they respectively develop is vastly different. Indeed some germs in fermenting develop scarcely any heat at all in comparison with the extraordinary 65° Fahr. evolved by the kôji. We all know, however, that in

the fermentation of horse-manure there is sufficient heat developed to raise the temperature of a conservatory several degrees above that of the external atmosphere.

Before concluding my remarks on pyrexia I have to call attention to a well-marked third cause, which is not, however, an independent one, but the result of a hybrid between the other two. I allude to the elevation of temperature which, during the first two or three days, follows upon severe injuries and surgical operations with lesions of external tissue continuity.

All operations are, as is known, followed by an elevation of bodily temperature. For even the element of pain itself is sufficient, in cases where there are no signs nor symptoms of shock-collapse, to slightly raise the temperature. Whereas after all severe injuries and operations, especially on the abdominal organs, accompanied by a solution of external tissue continuity, an elevation of temperature of from 3° to 10° may occur. Ay, it even occasionally happens that in some patients the thermometer will indicate an increase of 2, 4, 6, or even 8 degrees in as many hours. Now according to my ideas this elevation of temperature is due not solely to nerve influence, but to the action of germs also. And my reason for saying so is, that when operations are performed according to the antiseptic system of surgery introduced by

Lister, the elevation of temperature is almost invariably found to be much slighter than when no germicide is employed. Of course, while the antiseptic destroys the germs, and thereby prevents their adding to the bodily temperature, it has no effect upon the abnormal heat resulting from the nerve influence. Hence it cannot be expected that antiseptics, as some of their too sanguine votaries have asserted, ought to prevent the appearance of all pyrexial signs after operations. In fact it has been over and over again shown that in consequence, I suppose, of some human constitutions being peculiarly susceptible to its irritating toxic effects, the deadly germicide—the nervous-irritant poison carbolic acid —instead of invariably diminishing, actually in exceptional cases increases, the temperature of the patient's body; not, however, from failing to kill the germs, but from adding its nerve-irritating effects to those of the operation. Which fact is another good example of how necessary it is, in attempting the interpretation of abnormal biological phenomena, never in any case to restrict our lines of reasoning to purely practical medical facts and observations. For if we do we assuredly fall into errors, just as I observe Dr. Bantock has done (in his otherwise able paper in the last volume (lxiv) of the 'Medico-Chirurgical Transactions') when he comments unfavourably on the Listerian method, citing a clamp and a drainage case of

ovariotomy, in both of which the temperature only rose to 103·6°, while in four others performed under the influence of the carbolic acid spray it rose considerably higher—in one case which he cites actually to 107·2° Fahr. At the same time, however, he admits that on an average the Listerian method has at least the advantage of diminishing post-operative abnormal temperatures by four-tenths of a degree, as calculated on fifty-five cases, the average highest temperature being 100·3°, the lowest 99·9°. As is here seen, this beneficial effect of germicides after operations is but another link in my chain of reasoning regarding the important part played by germs in the production in the majority of cases of abnormally high temperature.

When the etiology of pyrexia is thus interpreted, it is easy to see how quinine and other germicides produce their effects in lowering the temperature of the body. They, simply by killing the germs, put a stop to the fermentative process upon which the rise of the bodily temperature of the patient depends. The action of aconite and digitalis, neither of which is known to be germicidal, is not so readily explained. They act, I believe, through their direct influence upon the circulation. Lowering the heart's action by diminishing its power, through the depressing effect they have on the nervous system, and by diminishing circulation they diminish tissue metamorphosis, and, as is well known, heat as well as functional

activity is one of the direct products of tissue oxidation.

Cold again, I think, acts in a threefold manner:

a. Like digitalis and aconite it diminishes tissue metamorphosis by its depressing action on the nervous system, and thus reduces the bodily temperature.

b. It diminishes the oxidation of organic substances by its direct cooling action retarding chemical affinity.

c. It is a true germicide. For cold kills germs just as it kills other animals and plants.

The chief action of germs, as we see, is fermentative, and we all know that every species of fermentation is hastened by moderate heat, and retarded by moderate cold. While extreme heat as well as extreme cold annihilates the process altogether. Cold, then, judiciously employed, ought to be a most powerful adjunct in the treatment of hepatic germ disease, and so it is, as we shall subsequently see. This of course is an entirely new view of the *modus operandi* of cold in the treatment of hepatic and other forms of pyrexial diseases; but although this is the first time I have put these ideas on paper, they have for many years past occupied my attention, and the more consideration I bestow upon them, the stronger is the hold they take of me.

Subacute Atrophy of the Liver (Rokitansky's Red Atrophy).

The true pathology of this condition of liver—at least as a separate form of disease—is as yet unknown. All that I can say on the subject is that in 1854, while a student at Vienna, I saw that Rokitansky gave the name of red atrophy to all cases of atrophied liver, the substance of which was more than usually red, from a hyperæmia of the hepatic capillaries induced apparently by the outward flow of blood from the portal veins being obstructed. The livers in persons so affected, and who, he said, had in general been the subjects of repeated attacks of remittent or intermittent fevers, were occasionally reduced to half their normal bulk. I was then, as I am still, very much inclined to doubt the propriety of giving to this state of liver the honour of being considered as a separate form of disease; for to me it appeared, and still appears, merely to occupy the debatable ground between the acute and chronic forms of hepatic atrophy, just at the very spot where it is exceedingly difficult to say where the one form of the disease ends and the other begins. In fact I look upon Rokitansky's so-called red atrophy as merely a less advanced stage, or a less pronounced form of virulent acute atrophy. For they differ in no way whatever from each other clinically except in so far as red atrophy runs a slower and

consequently a less severe course. Red atrophy lasting from six days to even six weeks, whereas acute or yellow atrophy may terminate fatally in six hours or at the longest within six days. Being thus less virulent, red atrophy is naturally enough less frequently fatal, and after death the liver presents only in a modified degree the pathological appearances of acute atrophy. Being red or yellowish-red and firmish, instead of a saffron hue. and pultaceous. As these are my own and as yet unsupported opinions, I shall not in order to prove them select an illustrative case of the disease from among those I have myself seen, and consequently might be suspected of having a personal interest in recording with partiality, but I shall take one from a totally independent observer, and he too one who, from the very title he gave to his case, had evidently himself some doubts as to which category of atrophies of the liver it properly belonged to. The case has likewise the additional advantage of being so fully as well as ably reported, that it admits at least of a decided if not even a correct conclusion being drawn from it. If I had adopted my old teacher Rokitansky's views, I should unhesitatingly have called it a case of red atrophy ; but as they are different from his, I think its reporter, Dr. Macnaughten Jones, has with great propriety cautiously and somewhat doubtingly entitled it 'A Case pre-

senting the Symptoms of Acute Yellow Atrophy of the Liver,' and given at the end of his paper the following nine reasons for so doing.

1. The sudden accession of the attack ; 2. The slight constitutional disturbance at its commencement ; 3. The cephalalgia ; 4. The obstinate bowel and intolerant stomach ; 5. The peculiar head-symptoms, and their rapid and intense occurrence ; and, as regards physical signs, 6. The diminished dulness over the liver ; 7. The tenderness also, though not excessive, yet well marked ; 8. The stools being most peculiar in occurring when they did, and being of a hæmorrhagic nature ; 9. The pulse, except on a few occasions, was from 75 to 80 per minute.

The following is an abstract of the history of the case as given by Dr. Macnaughten Jones in the 'British Medical Journal' May 4, 1872.

'The patient, a married woman aged 20, in the seventh month of her pregnancy, had her attention suddenly drawn to the jaundiced tint of her skin on October 18. She had been lately greatly depressed about her husband's health, having had a shock from seeing him spit up blood. When Dr. Jones saw her, she had intense pain in the head ; her pulse was normal. She was sitting up, and had been attending in her place of business that day up to the time when he was called. He ordered her to go to bed ; the next

morning he found the pulse natural, no heat of skin, an icteric tint marked over the body, and intense depression of spirits. On examining the liver it appeared smaller than usual. The bowels were costive, and she felt sick. A powder containing ten grains of nitrate of potash, three grains of James's powder, and three grains of grey powder, was administered at bedtime. In the middle of the night, labour came rapidly on, and she was delivered of a dead male child after a few labour pains. The next morning she had no pain or tenderness anywhere; the stomach was settled; the mind more cheerful; the pulse perfectly natural. The icterus, however, was rather increased than diminished. The stools passed up to this time were perfectly colourless, and the urine was thick and portery. A peculiar heavy odour was exhaled from the surface of her body; but there was almost unusual clearness over the region of the liver, and an impossibility of feeling the margin of the gland. The bowels not being moved on this day, an enema of oil and yolk of egg and gruel in the evening brought away two copious watery discharges.

'October 21st. She was greatly jaundiced. Her mind was wandering; she did not recognise her friends, and was delirious at times, muttering on religious subjects, fancying that a person beside her was Jesus Christ. She had had one small stool,

nearly white. The urine was scanty, thick, deep-coloured ; pulse 115. There was a heavy smell from the body. She lay sunk in the bed, with her eyes closed ; and was difficult to rouse. There was no pain or tenderness anywhere. The lochia were not suppressed, and of fair colour. She had not the least tympanites. There was unusual clearness still over the hypochondriac region. She took this day the same nutriment ; and a teaspoonful of brandy in milk and wine was added alternately every hour. A saline purgative was given ; and, as it did not act, the enema was repeated with assafœtida at night. Mustard stupes and large linseed cataplasm were alternately applied over the abdomen.

'On the 23rd she was still drowsy, but not so wandering as on the previous day. She had a draught of infusion of roses and sulphate of magnesia with spirit of chloroform, which operated. There was no change in the stools and urine. At night she took two pills containing eight grains of compound rhubarb pill, five grains of calomel, and two grains of extract of hyoscyamus. She had two stools in the night of the same character as before.

'On the 24th there was no sickness of stomach ; she was more conscious ; the pulse had fallen to 80. She spoke rationally.

'On the 25th she was much improved ; the icteric tint was less ; the urine of better colour. There was

slight secretion of milk. The head symptoms were entirely gone. The saline aperient draught was repeated. This brought down frequent copious stools of a watery consistence and of a dark hæmorrhagic nature. The lochia had passed in quantity again; and though there was great weakness and prostration, her general symptoms were much improved.'

From the 26th onwards, the stools and urine improved. Her strength gradually returned, and Dr. Jones ceased to attend her on November 25. When seen on February 24, she was in perfect health, and expressed herself 'never better in her life.'

This case I regard as a very good example of what Rokitansky called red atrophy of the liver, and I think was, as is seen, with perfect justice entitled by Dr. Macnaughten Jones 'A Case presenting the Symptoms of Acute Yellow Atrophy of the Liver,' the red, I believe, as already said, being nothing more or less than a mild form of the *yellow* atrophy of the organ, both as regards its clinical history and its pathology; and being such demands nothing more or less than a modification of its treatment.

In order to show how easy it is to give diseases wrong names, I quote the following case of subacute atrophy (red atrophy so called) of the liver, which appeared in the 'British Medical Journal' of November 6, 1880, under the misleading title of

Acute Atrophy of the Liver.—Mr. Cullingworth narrates the history of the case in a married lady, aged 28, which terminated fatally at the end of four weeks, as follows. The patient, who was nursing her first child, had an attack of jaundice, which, for the first three weeks, appeared to be of the mildest character and simply catarrhal. Severe symptoms suddenly supervened, ending in delirium, coma, and death. The temperature did not rise until the last twenty-four hours ; an hour before death it was 105° Fahr. The jaundice became intense, and hæmorrhage occurred. After death, the liver was found atrophied and softened, for the most part reddish in colour, with large irregularly distributed patches of orange-yellow. The weight of the organ was 677 *grammes* (about 1½ lbs.). Hæmorrhagic spots were found over the heart, the mesentery, and the large and small intestines. Microscopic examination showed destructive changes to have advanced further in the red portions of the liver than in the yellow ; in the former the liver-cells had entirely disappeared. Professor Arthur Gamgee had made a chemical examination of the liver, and of the whole of the urine passed during the last thirty-six hours of life. The amount of urea found in the urine was considerable ; and, while abundance of leucin and tyrosin was found in the liver, the urine contained no trace of either of these substances.

Two facts in the history of this case incontrovertibly show that it more properly belongs to the subacute than to the acute form of atrophy of the liver. Though it is one of those transition cases which possess some of the characteristics of both. 1st. The reddish colour of the tissues. 2nd. The duration of the disease, namely four weeks.

The third and last variety of atrophy of the liver was formerly called contracted or cirrhosed, but is now more appropriately known under the title of

Chronic Atrophy of the Liver.

This condition of the hepatic organ is, and I think most unnecessarily, very imperfectly understood by the great majority of practitioners. The misunderstanding arising, in great measure, from the number of different names that have been given by pathologists to the multitudinous mere varieties of one and identically the same disease. I shall now endeavour to unravel the tangled pathologico-clinical skein, and place the subject, as far as I am able, in a clear light.

Chronic atrophy of the liver has been named 'cirrhosed,' 'nutmeg,' hobnail,' 'contracted,' and 'dram-drinker's' liver, according to its supposed exciting causes and pathological appearances.

It has been most injudiciously called 'cirrhosed,' from the simple fact that neither the Latin word

cirrus, a curl or lock of hair, nor the Greek word *kirrhos*, signifying tawny, in the slightest degree defines the pathological condition of the liver which each of them is supposed to denote. Again, the term 'hobnailed' liver is applicable to only one special form of atrophy. While that of 'dram-drinker's' liver is still less appropriate; for, as I shall presently show, the most typical pathological forms of the so-called dram-drinker's liver are not only to be met with in the temperate adult's, but are even to be found in the milk- and water-imbibing babe's liver.

A chronically atrophied liver—that is to say one that has been hitherto described under the name of a 'contracted' or 'cirrhosed' liver—may indeed be, I believe, pre-natal; for as atrophy of the liver is, sooner or later, the inevitable sequel of obstruction of the common bile-duct, and an imperforate duct is oftentimes a congenital abnormality, I see no reason whatever to doubt the possibility of the existence of an atrophied liver in a new-born child. I know, indeed, for a fact, from my knowledge of the literature of infantile hepatic diseases, that children at the breast—from a week or two old and upwards—who have succumbed to jaundice the result of a congenital deficiency of the common bile or hepatic duct, have had livers not only atrophied, but truly cirrhotic in the widest sense of the word. In so far

at least as the whole secreting substance of the organ has been found interspersed with, and enclosed within, dense bands of hypertrophied and hardened fibrous tissue. While, curiously enough, in many of the well-marked cases the spleen has been found to be considerably hypertrophied, just as if it had been attempting in some way or another to do vicariously, in addition to its own normal work, the work of the liver. In precisely the same way as the spleen is sometimes found enlarged in cases of acute atrophy of the liver. In fact, a more or less distinctly atrophied condition of the liver may be said to follow upon all the various forms of chronic hepatitis, be the exciting cause of the inflammatory enlargement what it may.

The signs and symptoms which are in general indicative of a chronically atrophying liver are:—

Sallowness of countenance; yellowishness of the conjunctivæ; disordered digestion; loss of appetite; furred tongue; flatulence; irregular stools; dark urine; depressed spirits; diminished mental power, and general inability to exertion of any kind whatever. The liver itself is uncomfortable, but not painful; and smart percussion adds but little to the discomfort. The dull perpendicular area is reduced *pari passu* with the advance of the atrophying process, until it may be less than two inches in extent.

Intense jaundice is seldom or never seen in cases of chronic atrophy, except in those where it follows upon occlusion of the common bile-duct, in which case the discoloration may indeed be intense, sometimes amounting to an actual dark yellowish-green hue. There is also almost invariably a gradual decrease of the jaundice as the chronic atrophy of the liver advances, even in spite of there being persistent occlusion of the common bile-duct, as, for example, by a large gall-stone. This fact is well illustrated in the case of a woman aged 60, recorded in vol. xv. of the Pathological Society's 'Transactions' by Mr. Wale Hicks, in which it is stated that the jaundice decreased, 'until at last the skin was free from any icteroid tinge.'

Ascites is a common accompaniment of cases of chronically contracting liver, and it is easily explained by the gradually atrophying glandular tissue contracting round the large portal vein and vena cava inferior, and compressing them to such an extent as to impede the outward and upward flow of the abdominal venous blood.

Along with the ascites there is marked dilatation of the external abdominal veins. When the ascites is considerable, it, in its turn, impedes the flow of blood through the iliac veins, and thus produces œdema of the lower extremities.

In the earlier stages of the contraction of the

liver tissue, ascites is absent; but in the later, never.

Hæmorrhage from the bowels is nothing unusual in chronically atrophied livers. At the Medical Society of Dublin (1881) Dr. Quinlan exhibited the liver of a patient who died in St. Vincent's Hospital. The patient was a light-weight steeplechase-rider, aged 44. He was of most temperate habits. About two years previously he got a very severe fall, which was followed by an attack of what Dr. Quinlan inferred to be inflammation of the liver. He died from intestinal hæmorrhage. On examination of his body, a 'hobnailed' liver was found. Its weight was only 2 lbs. 2 ozs. There were no signs of syphilis, cardiac disease, or albuminuria; but the patient had been very deeply jaundiced.

In December 1881, I was summoned to Grantham, to meet Dr. Paterson in consultation on a suspected case of this kind, occurring in a young lady of 14; but after careful examination we came to the conclusion that the hæmorrhage did not come from the diseased liver, but from a gall-stone ulcerating its way into the intestines. She was treated accordingly, and the hæmorrhage did not recur, as it would have been sure to do had it been the result of hepatic atrophy. The form of passive hæmorrhage associated with an atrophied liver is, as a rule, best treated with repeated doses of a mixture like the one of which the formula is given at p. 266.

The sequel of this case may be judged of by the following quotation from a letter I received from Dr. Paterson on January 25, 1882, in which he says :—

'I thought you would like to hear how Miss—— progresses, and I think I can give you a pretty good account of her. She has apparently quite regained her strength, and appears quite well, with the exception of a quick pulse, and that, I believe, she always has. She has regained her flesh. Appetite good, sleep natural, bowels regular, and stools of proper colour.'

The most common cause of atrophied liver, whether in the child or in the adult, is obstruction to the flow of bile into the intestines, from a more or less permanent occlusion of the ductus communis choledochus. And what is equally noteworthy is the fact that one and all of the various forms of chronic atrophy (which I shall presently describe) are invariably preceded by a proportionate stage of hepatic inflammatory or congestive enlargement of the organ. The stage of enlargement may be long, or it may be short; but, in every case of chronic hepatic atrophy, it must have had a preceding existence. The brevity of the stage of enlargement in some cases may be conjectured from the fact that Dr. Legg found the livers of animals in a markedly atrophied (which he calls cirrhotic) condition four-

teen days after he had applied a ligature to their common bile-ducts.[1]

Were this merely a pathological instead of a clinical treatise on diseases of the liver, I should devote considerable space to the microscopic appearances found in all cases of chronic atrophy; but being as it is a practical treatise, I must refer those of my readers who take any special interest in this subject to an admirable paper on the 'Histology of the so-called Nutmeg Liver,' by Dr. Wickham Legg, in vol. lvii. of the 'Medico-Chirurgical Transactions,' where they will find the gist of the matter clearly and fully put forward; likewise to a good account of the literature (as well as to the morbid anatomy) of what is called hypertrophic cirrhosis of the liver, which has been given by Dr. Robert Saundby in vol. xxx. of the 'Pathology Society's Transactions.' To this paper I would also refer the reader who is specially interested in the subject of the multiplication of biliary canaliculi in hypertrophic cirrhosis of the liver, as it will not do for me to go into the matter here, seeing that it has nothing to do either with the diagnosis or treatment of such cases.

I shall now proceed to point out the pathological varieties of chronic atrophy of the liver, in so far at least as a knowledge of them is of importance to us from a clinical point of view. And to begin with, I

[1] *St. Bartholomew's Hospital Reports*, 1873.

must of course take the form which, as has just been said, is most common. Namely, the one met with in this and in all temperate climates, where, as a direct consequence of the atrophying process being universal in extent and uniform in degree, the organ, though it may become exceedingly small and dense, yet retains its relative proportions. Cases of this kind frequently follow upon the congestive form of hepatitis induced by a permanent impediment existing to the flow of bile through the common bile-duct into the duodenum. Such, for example, as occurs when the intestinal orifice of the bile-duct is sealed up by a cicatrised ulcer. The continued backward pressure of the accumulated and long pent-up bile causing such an amount of disturbance in the capillary circulation as is sufficient to induce a degeneration and ultimate shrinking of all the hepatic tissues, and, from the fact of the pressure being exerted universally and equally upon the intercellular tissues and the secreting cells, the atrophying process is so nearly uniform that there is a shrinking of the whole tissues of the organ, and no hobnailed appearance is perceptible in uncomplicated cases.

As I give a most typical example of this pathological condition of uncomplicated true chronic atrophy when specially speaking of the value of physiological chemistry in connection with the diagnosis and treatment of obscure hepatic dis-

case, I shall not introduce it here, but merely refer the reader to page 719. I may here add that as in acute and subacute, so also in chronic atrophy of the liver both tyrosin and leucin are detected in the urine in the last stages of the disease. A fact well worthy of the attention of the clinical physician in the study of obscure hepatic cases. In cases where a syphilitic taint is associated with the chronic hepatic atrophying process the contraction of the tissues sometimes occurs irregularly. In consequence of the erratic deposition in different parts of the organ of the syphilitic materials, either of the so-called gummatous or of the fibroid forms. While again, in cases of perihepatitis, where only limited portions of the liver have suffered from inflammation, they alone are the parts that undergo the atrophying process, and consequently at the *post-mortem* examination the organ presents a peculiarly irregular shape, on account of its tissues being more atrophied in one direction than in another.

In all cases of chronic hepatic atrophy, however, be the cause what it may, the fibrous capsule of the liver is in general not only found thickened, but at the same time firmly adherent to the hepatic tissue. I must now return for a moment or two to the dram-drinker's liver, for there actually exists a peculiar pathological condition of the liver which may be said to be the especial possession of the habitual tippler

of ardent spirits, which hence obtained the expressive, though only vernacular, name of dram-drinker's liver. More especially in those cases where the tipple indulged in has been Jamaica rum, and particularly so when it has been taken in a hot climate, the condition of liver has an exceptional right to be christened, after its most characteristic appearance, 'hobnail,' as it decidedly looks as if it were all studded over with hobnails. This nodulated so-called 'hobnail' appearance of the external free surface of the liver is due, not to a series of local and limited portions of the organ becoming atrophied, as in the cases of perihepatitis just alluded to, but to a definite and distinct irregularity occurring in the shrinking of the different tissues of which the organ as a whole is composed. The fibrous parts, or more correctly, histologically speaking, the intercellular (now hypertrophied) connective tissue, shrinking not only more rapidly, but more completely, than the secreting hepatic cells, small lobuli are thereby formed in consequence of the puckering in, as it were, of their surrounding connective tissue, and cause the surface of the organ to assume a distinctly nodulated appearance. Not at all unlike the surface of an old-fashioned church oak door thickly studded over with globular-topped hobnails. A wax model of a magnificent specimen of this kind of liver exists in University College Anatomical Museum.

That this morbid anatomical appearance should come about after a preceding inflammatory enlargement of the organ is easy enough to understand, when it is remembered that during the inflammatory stage the connective tissue becomes greatly hypertrophied, and, by the pressure it then exerts on the secreting cells, interferes not only with its own but at the same time with their proper nourishment, and thereby induces degeneration of tissue. So that ultimately both the size and numerical proportion of the secreting cells to their surrounding, now dense areolar tissue, are so much altered that the normal smooth uniform appearance of the surface of the organ is entirely lost, and in its place is substituted the abnormal condition to which has been applied the term hobnail liver. But that neither hobnail liver nor any other form of shrunken, cirrhosed, or atrophied liver, is always due to dram-drinking, may be inferred from the fact that even typical examples of this peculiar pathological condition are occasionally, as I previously said, though very rarely, met with in children. One of the very best examples of a hobnail liver occurring in a child, that I am acquainted with, is described by Dr. Wilks. It occurred in a boy aged 11, who had been an out-patient at St. Bartholomew's Hospital in 1862. The boy's necropsy was carefully made by Dr. Wilks, and he describes the condition of the liver as follows :—

'The organ appeared to be of the usual size, but its surface was studded with nodules about the size of peas, which at first sight appeared like little tumours growing on the organ. On section the same appearance was found throughout the liver. On closer examination the disease was seen to be cirrhosis.'[1] The boy was the son of an itinerant chair-maker, and was said to have occasionally participated in the gin and water which his father indulged in; but that is not sufficient evidence to make it appear at all probable that he was a confirmed youthful tippler.

In fact, general atrophy of the liver may occur at any period of life when there exists an impediment to the free exit of bile from the secreting hepatic cells. Dr. Quain exhibited in 1854 to the Pathological Society a specimen of lobulated liver taken from a boy 3 years and 10 months old, which looked like a lobulated kidney. The whole organ, with the gall-bladder attached, weighed only 13 ounces. Its capsule was opaque and thickened, and the sides of the lobules were adherent to each other by old inflammatory adhesions. In the bottom of each fissure between the lobules was a branch of the portal vein. The child died with the symptoms of general anasarca, from the combined action of renal and hepatic disease. The two kidneys weighed together 6 ounces. Their tubules were full of casts of oily epithelium. The

[1] *Pathological Society's Transactions*, vol. xiv. p. 175.

urine had been albuminous. There was no jaundice.

A still more curious example of chronic atrophy of the liver occurred in a girl aged 13, who died in University College Hospital in 1856. For the organ looked much more like a brain than a liver, not only in colour but in the way it was convoluted and lobulated, while, strange to say, it was actually divided by one great fissure into two slightly unequal hemispheres. The girl died within twenty-four hours after her admission into the hospital, and, from her being too ill to give a history of herself, little is known of the case; but Dr. Hillier, who made the necropsy along with me, from finding the stomach full of black and clotted blood, coupled with the peculiar condition of the liver (which weighed 26 ounces), entered it in the hospital books as one of 'cirrhosis of the liver, accompanied with profuse hæmorrhage into the stomach and intestines.'

To show that an atrophied liver is not in the least degree uncommon in youth, not only without there having been an habitual indulgence in ardent spirits, but even without any other distinctly assignable cause, I may mention that Sappey has described a number of cases to which he gave the special title of 'congenital cirrhosis of the liver.' Moreover, Dr. Moxon has recorded an extreme case of the kind which occurred in a young and temperate man of 20

years of age. In this case the spleen was greatly enlarged, and a cluster of veins (*caput Medusæ*) developed near the umbilicus. From the veins bleeding occurred on one occasion, and fatal hæmatemesis was the final symptom. The spleen weighed 36 ounces. A large vein ran from the right branch of the vena portæ along the ligamentum teres towards the umbilicus, probably the permanently patent umbilical vein. The case was thought by Dr. Moxon to be one of congenital cirrhosis followed by perihepatitis.

There is yet another form of chronic atrophy of the liver, which by several writers has had the special term of syphilitic prefixed to it; but some people are so very fond of making subdivisions of all varieties of morbid conditions now-a-days, that I think it is scarcely worth while to do more than call attention to the name. For just as it is fashionable for us to differentiate during life cases of gouty bones, gouty brains, gouty stomachs, and gouty everythings, so it is thought by some to be equally correct to apply the terms syphilitic cirrhosis and syphilitic cancer even to simple cases of cirrhosis and of cancer in which the morbid taint of the syphilitic poison chances to be accidentally present. There is, however, no doubt a form of disease of the liver which well merits the cognomen of syphilitic, as it possesses special and well-defined features of its own, and I shall give special consideration to it at the proper time; but

at present I dismiss the syphilitic cirrhosis as a mere variety of a pathological condition unworthy of special consideration in a practical clinical treatise such as this pretends to be.

Having thus run through the most salient characters of the various forms of chronic hepatic atrophy which have been separately described by different writers both at home and abroad, I shall now give an illustration of what may be called the ordinary form of the disease met with in this country. The case I shall select for my illustration is one which I several times saw in consultation along with Dr. Bannister of Addison Terrace. It is such a typical example of the commonest form of chronic atrophy, both as regards clinical history and mode of fatal termination, that I shall relate it somewhat fully.

On September 28, 1879, I was called to meet Dr. Bannister at the patient's house at Shepherd's Bush, and there I saw a stockbroker of 38 years of age, who had been for several months previously under Dr. Bannister's care. The history given to me was, that, notwithstanding his having been what is usually denominated a free liver and hard drinker for many years, he had enjoyed, comparatively speaking, good health until the summer of 1878, when he was attacked with jaundice from acute hepatitis. After the inflammation of the liver subsided, the organ speedily returned to its natural size. It did

not, however, long stop there, but went on gradually diminishing, and the jaundiced tint, though much faded from what it was at first, never entirely disappeared. In the next July, 1879, he had a second attack of hepatitis, and rapidly became of a deep jaundiced colour. The liver, though painful on this occasion, never perceptibly increased in size—at least if it did it was only to a small extent. The hepatitis of this second attack soon yielded to remedies, but the jaundiced hue of the skin remained persistent. A week or two before I was called in, the hepatic dulness began to decrease in extent, and concomitantly with the diminution in the size of the liver, ascites set in. When I first saw the patient—which was about four months after the commencement of the second attack of hepatitis—the liver had already become so shrunken that its perpendicular nipple-line dulness was barely two inches. The abdomen was at the same time enormously distended with fluid, and the superficial veins all over it greatly dilated. Thus clearly indicating that the ascending vena cava was already severely constricted by the contracted liver. The patient's legs were at the same time œdematous, and the prepuce and scrotum similarly affected. The conjunctivæ were greenish-yellow, and the skin of a deep greenish-yellow jaundiced hue. The pulse was 120 and feeble. The bowels open (three or four times a day). The urine scanty and

deeply bile-coloured, with a copious red brick-dust urate-looking deposit. He had been slightly delirious during the night preceding the day on which I first saw him, and consequently the case was regarded as a very unfavourable one. He was ordered to be tapped, and to have ten grains of James's and five of Dover's powder given to him at bedtime. Three days later I again saw him. Dr. Bannister had tapped him two days previously, and had drawn off no less than 28½ pints (3½ gallons) of a rich saffron-coloured serum, so impregnated with bile pigment as to stain his hands of a bright orange-yellow tint. After the fluid was evacuated the patient rapidly rallied and greatly improved. His pulse became stronger and less rapid, though it remained still close upon 100. The urine continued to be very scanty, only ten ounces in the twenty-four hours, and loaded with bile and brick-red lithates. The abdomen in forty-eight hours had refilled itself very nearly to the same extent as it was before. As the kidneys were acting badly, a mixture of squills, digitalis, nitrate of potash, in a decoction of broom tops, was prescribed, and he was ordered again to be tapped, and this time a drainage-tube to be inserted. At this second tapping (made six days after the former, and with a fine drainage trocar), 2½ gallons came away in the first twenty-four hours, 1¾ gallon in the second, and ¾ gallon in the third. Unfortu-

nately the trocar got accidentally stopped up, and had to be withdrawn, and when I again saw him, thirty-six hours afterwards (on October 7), the abdomen was about half as much filled as it was when I saw him on the first occasion. Fortunately the kidneys were now acting much better. Instead of passing only ten, he was now passing about thirty ounces of urine in the twenty-four hours. The urine was still, however, very dark in colour and loaded with lithates of a strikingly deep red hue. When they were separated by filtration, washed with distilled water, and dried, they looked exactly like red-lead powder. From the 8th of the month the quantity of urine passed each day was carefully noted, and it was found that

On	the	8th	was passed	30 ounces.
,,		9th	,,	$27\frac{1}{2}$,,
,,		10th	,,	$27\frac{3}{4}$,,
,,		11th	,,	30 ,,
,,		12th	,,	34 ,,
,,		13th	,,	$34\frac{3}{4}$,,
,,		14th	,,	40 ,,

The abdomen having again become entirely filled up with fluid, the drainage-tube was reinserted on the 12th, but on the left instead of on the right side of the abdomen, as the patient said he preferred to lie on the latter side. In twenty-four hours no

less than other 460 ounces (eleven quarts and one pint, or nearly three gallons) of peritoneal secretion had again flowed away. It then suddenly ceased to flow, and on the three succeeding days none whatever came away. While on the fourth day it began to flow again, and before it again entirely ceased between one and two quarts came away. No sooner did the flow cease than the urine again became very scanty. So much so indeed, that on the day preceding the patient's death, which occurred on October 18, that is to say three weeks after I first saw him, only four ounces of urine were passed. During the last few days of his life he was more or less delirious, no doubt from uræmic poisoning in consequence of the kidneys having struck work, and about twelve hours before his death he gradually sank into a comatose stupor.

Here it is seen that in this patient the enormous amount of twelve gallons of ascitic fluid was secreted by the peritoneal cavity in thirteen days, giving on an average close upon a gallon a day, all of which fluid was withdrawn by tapping within the same length of time. The marvel is how the serous peritoneal membrane could secrete it so rapidly from the blood, neither food nor drink having been taken in sufficient quantity to supply that amount of fluid. There is but one way to account for it, namely, that this large amount of fluid was extracted by the lungs in the

shape of gasiform moisture from the inspired air, added to which the aqueous vapour normally generated in the tissues of the body during the process of the transformation and assimilation of the hydrocarbons supplied by the food was taken up by the peritoneum instead of extracted from the blood by the kidneys.

Unfortunately no necropsy of this interesting case was obtainable.

Treatment.—In the treatment of all cases in which an atrophying liver is diagnosed, it is not so much the morbid condition of the organ itself as the original exciting cause of the pathological state which is to be attacked, and only the most prominent of the symptoms it induces treated. Whereas, again, when the organ is already decidedly atrophied before the patient comes under the practitioner's care, no matter whether the contracted state of the liver be the result of dram-drinking, malarial hepatitis, obstruction to the outward flow of bile into the intestines by a gallstone impacted in the common bile-duct, or any other cause, regulated diet and regimen are the chief means which the enlightened practitioner adopts to retard the progress of the disease and prolong the life of his patient. To aid in that object, the following suggestions, both as regards food and medicine, may be found not unacceptable.

A. 1st. Give a due proportion of animal and

vegetable food. The former *always fresh*, and rather underdone. The latter especially of the starchy variety and thoroughly cooked. A good combination is milk and eggs along with tapioca, corn flour, ground rice, sago, or arrowroot, in the shape of puddings.

2nd. Moderate and slow exercise in the open air; sleeping in a high altitude, and, when the weather permits of it, with the bedroom window open.

3rd. The careful avoidance of cold baths, or any other sudden change of temperature which will give a shock to the system, is also a point of vital importance.

4th. Night and morning brisk cutaneous frictions with hair flesh-brushes.

B. While as regards medicines—1st. Stomachic tonics and vegetable purgatives are to be used according to circumstances. Mineral acids and ferruginous salts I have ceased to employ, never having seen the slightest benefit follow their administration; while, on the other hand, the constipating effects they invariably produce upon the bowels always require to be counteracted by other medicines, otherwise hepatic congestion is greatly favoured.

2nd. Symptoms of ascites and œdema are to be counteracted in the usual way—relieved by tapping, if necessary. One of the best forms of diuretic I have found in these cases has been a combination of digitalis, squills, nitrate of potash, and sweet spirits of nitre in an infusion of broom tops, and a dose of

it given every six hours. For further remarks see chapter on Ascites, at page 1044.

3rd. In cases with a syphilitic history, I add to the above 5 grains or so of iodide of potassium per dose. Small doses of iodide of potassium—that is to say, anything under $1\frac{1}{2}$ grain—I find most decidedly objectionable in bringing on much too rapidly the disagreeable weakening symptoms of iodism—to wit, deafness, running at the nose, pimples on the face and back, &c. &c.

Pyæmic and Septicæmic Forms of Jaundice.

As ichoræmia, or the purulent diathesis, may be said to be the pathological synonym of pyæmia (for they equally belong to the same morbid state of system as is included under the general term of *Metastatic Dyscrasiæ*), and pyogenic or putrid fevers may be equally regarded as the pathological synonyms of septicæmia, in the remarks I am now about to make on pyæmic and septicæmic jaundice I include in them the morbid states usually signified by the other names above mentioned. Only making the broad pathological distinction, which I believe (notwithstanding what has been affirmed to the contrary) is a correct one, that while the formation of multiple abscesses is the rule in the pyæmic, it is the exception in the septicæmic group of these morbid

states. My object in considering them together is to save space; and there is no harm in doing so, seeing that they all belong, as regards their etiology, to the same class, which happens too to be identically the same as that to which belong the contagious jaundice of the tropics and the jaundice of sporadic acute atrophy of the liver in temperate zones. The speaking of them conjointly is, moreover, all the more permissible from the fact that it matters not one iota, from a pathological point of view, whether the disease-germ spawn of the specific form of the jaundice has been introduced from without, as in the contagious jaundice of the tropics, or been generated within or upon some merely local part of the affected individual himself, as in pyæmia, by a morbid retrograde metamorphosis of decomposing tissues or secretions. For in all these cases the course of the specific pyæmic and septicæmic disease-germs during their career in the human body, in so far as their mode of action in producing the jaundice is concerned, is identical—the only difference being as regards their origin and primary modes of attack.

It has long been noticed that many of the otherwise common forms of both pyæmia and septicæmia are very frequently associated with a more or less well-marked yellow condition of the skin. In many cases the skin is sufficiently deeply tinted to merit being called jaundiced, while in others again it presents

more the characteristics of a chlorotic sallowness than of a true yellow hue. Moreover, in both sets of cases it not unfrequently happens that neither is the urine loaded with bile nor are the stools of a decidedly pipe-clay colour; while in others again, not only are the stools of a pipeclay colour, but the urine, besides being loaded with bile-pigment, is distinctly albuminous. Just as occasionally happens in cases where a jaundiced condition of the skin is associated with fevers, such as those of a typhus, typhoid, relapsing, and scarlet fever type; which pyæmic jaundice in the majority of cases still further resembles, from its being also associated with grave constitutional symptoms of a more or less febrile, typhoid, and hectic character; the skin being hot and dry, occasionally with petechiæ upon it; the tongue brown, dry, and crusted; the breath offensive; the lips covered with sordes. In the worst forms the urine is suppressed, the stools hæmorrhagic, the brain function greatly disturbed, and in general convulsions or coma close the scene, just as in contagious jaundice and acute yellow atrophy of the liver.

Septicæmic jaundice may be equally appropriately said to be the counterpart of pyæmic jaundice. For not only are its etiology and its symptoms, but even its chemistry, the same. Being, in my opinion, due to albuminoid fermentation, caused by the multitudinous development of protoplasmic animal organisms, be

their kind or name what it may. For although our present knowledge is insufficient to determine, either in pyæmia or septicæmia, their true nature, or indeed so much as the exact class in animated creation to which the ferment-organisms producing these different diseases properly belong, or even whether the deleterious action of the disease-germs on the human being is due to a chemical or to a morphological form of poisoning, I think we at least now know that the process by which they both act is as truly a fermentative one as is that of the saccharine fermentation produced by the multitudinous development of the spores of the torula or yeast plant in the barrel of beer or hogshead of wine. I even venture to opine that if this theory be extended into the domain of all forms of disease associated with increased temperature—be they fevers or be they inflammations—a clue to their true pathology is not unlikely soon to be found. I make this statement with all the more confidence, seeing that the analogous one (I made in a paper read before the Physico-Medical Society of Würzburg,[1] and which obtained for me the honour of its corresponding membership) that not only are the organic constituents of the animal and vegetable kingdoms identical as regards the chemical constitution of their oleaginous and albuminous principles, as

[1] 'Ueber Urohæmatin und seine Verbindung mit animalischem Harze.' *Verhandl. der phys.-med. Gesellschaft zu Würzburg*, Bd. v. 1854.

had been previously well known, but even as regards their pigmentary constituents, has been completely confirmed by the subsequent researches of numerous observers.

These remarks lead me to make one other observation on JAUNDICE AS A COMPLICATION OF OTHER FORMS OF GERM DISEASES, from its being a common though erroneous notion that the jaundice in the case of fevers is a mere accidental independent concomitant of the pyrexial state, and not, as it really is, part and parcel of the disease itself, quite as much as is the rash of scarlet fever or the pustules in small-pox. True jaundice, besides being almost an essential in all germ diseases of an epizootic as well as of a vegetable malarial type, both of the intermittent and remittent varieties, is not unfrequently met with in cases of typhus, typhoid, plague, and relapsing fever, or, as it is vernacularly called, famine fever. The reason of this is not far to seek, when we recall to mind the great tendency all disease-germs have to attack glandular organs in general, and the liver in particular.

Treatment.

The presence of jaundice in cases of pyæmia, septicæmia, or any case of fever or blood poisoning whatever, is always to be regarded as a most formidable complication. For experience has taught us that it not only necessitates a special line of

treatment of its own, but seriously embarrasses the treatment of the disease with which it is associated. And frequently, in spite of the greatest care, even when jaundice is present only in a slight form, the patient slips through the practitioner's fingers almost before he has become conscious of the serious nature of the case. This is not in the least surprising, seeing that before jaundice makes its appearance in those cases, the constitution of the patient has, in general, been so thoroughly undermined, as it were, by the primary disease, that therapeutical substances, the ordinary action of which would be beneficial, seem to be transformed into banes instead of antidotes, in consequence of the vital powers of the patient being so low that a trifling over-action of the remedy is sometimes sufficient to extinguish the flickering flame of life. Were proof wanting of the pertinence of this statement, it is abundantly furnished to us by the effects of the ordinary cholagogue cathartics. For while they may in most cases of non-germ jaundice be administered with advantage, and in all cases, it may be said, with perfect impunity, their employment is sometimes here followed by disastrous results. This arises from the fact of the vital stamina of the patients being so low in some of these cases that they sink under the exhausting effects of an excessive biliary secretion, when it is coupled with active purgation. Great care, as well as judgment, is

therefore necessary in the selection of the cholagogue cathartic by whose action the stomach and bowels are intended to be cleared out; for although it may be requisite to encourage, it is at the same time essential to control, the biliary secretion. For an excessive action in some of these cases is quite as detrimental to the welfare of the patient as a complete stoppage. Caution is therefore necessary in order to avoid the risk of letting the patient fall between these two stools. If a mercurial be deemed necessary, as probably it may be, unless the patient's vital powers seem good it must be given in the form of mild grey powder. But no matter what the cholagogue cathartic employed may be, no sooner has the alimentary canal been unloaded than it is advisable to administer germicides of a tonic character. And the best of these are quinine and its congeners. While, if the patient is considered sufficiently strong, salicylic, benzoic, and carbolic acid may be substituted. Always bearing in mind that in the complicated forms of disease now under consideration a remedy which may be given, even in a considerable quantity, in an uncomplicated case of such a disease is not only not always tolerated in similar doses, but may in small doses be found to be unsuitable or even detrimental. For general rules of treatment, see the chapter specially devoted to remedies and their modes of administration.

Cases of jaundice associated with septicæmia and pyæmia, in as far as the constitutional symptoms are concerned, are to be treated perfectly alike as regards the administration of quinine and salicylic, benzoic, or carbolic acids, which may be regarded as sheet-anchors. While the local signs and symptoms of each are to be subdued by its own appropriate remedies. For example, head symptoms, with an ice-cap, which has the double advantage of diminishing cerebral symptoms and reducing the whole bodily temperature by cooling down the blood as it circulates in the head beneath it. To tender parts and suppurations apply warm soothing applications, with poultices. Attend carefully to the action of both bowels and kidneys; and see that the patient has plenty of fresh air, and good, easily digested food, in small quantities at a time, but often. Agreeable companionship, and a cleanly kept sick-chamber, devoid of noise or much light. When the septicæmia is the result of a dissection-wound, or occurs in a puerperal, erysipelatous, or peritonitis case, promptitude in treatment is the element of success. For the whole course of even a fatal attack may be of but a few hours' duration. Early and deep incisions through the swollen parts, even before the appearance of suppuration, ought to be had recourse to. Leeching and hot fomentations, quinine and mineral acids.

Whenever purpuric (hæmorrhagic) blotches appear on the skin in a case of jaundice the result of germ disease, the attack is to be regarded as a grave one. No matter whether the jaundice be associated with typhus, typhoid, scarlet fever, or anything else. If the purpura be associated with much fœtor of the perspiration, the sufferer seldom recovers. Even although he gets apparently convalescent for a time, the chances are that, sooner or later, a relapse or sudden prostration will supervene, and a fatal termination is then much more likely than a recovery to be the result.

Violent sickness and diarrhœa are also most unfavourable signs in the course of jaundice the result of germ disease; almost as bad as that of severe and acute delirium.

As in the whole group of cases of hepatic germ diseases, both of the animal epizootic and the vegetable miasmatic malarial types, which we have just had under consideration, there is almost invariably, at some period or another of their course, a strong offensive odour emitted from the patient's excretions, not only from those of the bowels, but from those of the skin, kidneys, and lungs, and the significance of the effluvia met with in disease in general is but little appreciated and still less understood, I think it may be just as well for me here to devote a special chapter to its consideration, making

my remarks on the etiology and pathology of the smells emitted in hepatic diseases equally applicable to the fœtors of disease in general, in the same broad way as I treated the subjects of delirium and pyrexia.

The Fœtor of Disease.

All of my readers who have seen much practice must have been struck with the peculiarly sickening odour of the breath in cases of purulent phthisis and gangrene of the lung. They must likewise have noticed the offensive odour of the sweat in very many forms of disease, and also the peculiar stench of the stools and urine, not only in ordinary cases of jaundice, but more especially in those associated with pyæmic and septicæmic forms of blood-poisoning. In fact, in all cases where a deranged liver is the accompaniment of any form whatever of pythogenic affection. The very word 'pythogenic,' indeed, of itself signifies 'born of putrescence;' and, unless they possess particularly obtuse olfactory nerves, they may have occasionally felt called upon to exert a strong effort of moral control to prevent themselves beating a precipitate retreat from the obnoxious stench of sick-rooms where patients' erysipelatous sores have assumed a gangrenous character. With these introductory remarks, I shall now proceed to show

that the fœtor of certain forms of disease almost deserves to be ranked amongst the so-called signs of morbid action. My reason for saying so is that the effluvia emanating from the breath, the sweat, the eruptions, the urine, and the fæces are not only *sui generis*, but, in many cases, quite peculiar to and characteristic of the particular disease under which the patient labours. For example, the odour of the sweat in contagious jaundice, in the jaundice of acute atrophy of the liver, in malarial hepatitis, as well as in all the various forms of the jaundice complicated with pyæmia and septicæmia, is peculiar. Just as in typhus, typhoid, puerperal, and rheumatic fevers, it may almost be said to be of itself typical of the diseased state under which the patient labours. Everyone knows the sour odour of the perspiration in cases of rheumatic fever, the sweet vinous odour of it in delirium tremens; and I think that no one who has ever stood by the bedside of a case of acute glanders in the human subject will fail to remember the peculiar smell which proceeded from the patient's body, if he happened to be, as is more than likely, perspiring profusely. While the odour of the sweat in cases of hectic and pyæmia, though a less formidable nasal offender, must have equally succeeded in making an indelible impression upon his memory. Some describe the smell from pyæmic cases as resembling that of new-made hay, and that proceed-

ing from septicæmic patients like that of putrefying albumen. While others again have spoken of both as reminding them of different degrees of the mitigated stench of an old cesspool. This diversity of definition is not to be wondered at, seeing that every one of these odours is exceedingly difficult to describe in words, notwithstanding that they are quite characteristic and peculiar, and make such a deep impression on the mind that they are almost certain to be recognised again even on the very threshold of the sick-chamber. So difficult is it found to be to reduce to words some of the sensations produced by the smells emanating from the diseased human body, that the common and well-known odour of relapsing fever has been described by one author as a 'compound nondescript semi-fœtid mawkish smell.' No single word being capable of symbolising the idea desired to be conveyed to the mind of the reader.

The odours proceeding from sloughing cancers, phagedænic erysipelas, necrosed bones, and gangrenous tissues, which are reckoned by hospital nurses as olfactory abominations, are quite characteristic of each form of affection alluded to. Indeed, so much attention do nurses give to some of these noxious odours that it has been said, and I believe too not without good reason, that some of the nurses in small-pox hospitals can calculate with very considerable exactitude the virulence of an attack, both as regards its

danger to the lives of the patients and the risk of infection to the persons coming into contact with them, from the intensity of the smell alone. This is most probably true, seeing that the intensity of the stench is in direct proportion to the number, size, and condition of the suppurating pustules, coupled, as I shall presently attempt to prove, with the fact that the odour is in no case the direct product of the patient's tissues, but only of the disease-organisms infesting them. No wonder then that the fatality of the disease may be computed by the intensity of its odour. Typhus fever is equally thought by many hospital physicians to be most powerfully infectious when the pungent odour from the skin is strongest, which is from the end of the first week of the attack until a day or two after the crisis occurs and convalescence sets in.

The fœtor of hospital gangrene is almost equally prognostic as in small-pox and typhus. For not only the gravity of the condition of the individual patient, but his power of spreading the disease, may be reckoned by the intensity of the stench proceeding from his body. The effluvium from the mouth and nostrils of patients labouring under degenerative diseases of the respiratory passages, such as gangrene and acute tubercular softening of the pulmonary tissue, as also from malignant sore throat, syphilitic and scarlatinal ulcerated throats, diphtheria, &c., is, I

need only remind the reader, quite peculiar to each of these different forms of disease.

In cases of local gangrenes occurring in the course of germ diseases, such as typhus, small-pox, &c., the perspiration has a most peculiar sort of cadaveric odour, quite different from that of the mortifying part itself, but nearly as offensive, except when it is the lung which is mortifying, in which case the stench of the breath far exceeds that of the perspiration.

That the odours of all diseases do not communicate infection, notwithstanding that they may be peculiarly offensive and strong, was proved to me by one of my patients—an intelligent banker from Buenos Ayres. Who told me that during a fearful epidemic of yellow fever (contagious jaundice) which occurred while he was living there, he knew for a fact that not one of the men living at the cemetery—three miles distant from the town—and who were daily and almost hourly engaged in burying the dead, took the disease, although they were being continually exposed to the noxious smells proceeding from the corpses in the badly made coffins, which allowed the offensive effluvia to escape from them through their ill-constructed joints. My patient even said he had himself seen these men deliberately sitting on the coffins smoking their pipes, although the odour was so strong and offensive that it made him feel quite sick to be within a few yards of them. This fact seems to

show, first, that all disease odours are not infectious, and, secondly, that yellow fever, though, as is well known, it is infectious as well as contagious, is nevertheless not communicable through the mucous membrane of the air-passages, however readily it may be so through that of the digestive canal. While, thirdly, it further shows that, although in many cases the intensity of the bad smell from germs is an index of their virulence, it is not by means of the odoriferous principles of the germs alone that infection is communicated. To make this perfectly intelligible to my readers, after having so distinctly shown that the intensity of the stench is in many cases of germ disease an index of virulence, I shall relate the case of a medical friend, physician to one of our south coast hospitals, who, on calling one day upon me at University College, no sooner entered my private laboratory than he beat a sudden retreat, exclaiming 'Oh ! you have prussic acid! I can't stand it. The smell makes me so ill.' To me the odour was imperceptible. It proceeded from a stoppered bottle in one of the cupboards. The servant was ordered to remove the offending bottle into another room, and my susceptible friend entered. He then told me some curious details about the effects the odour of prussic acid had upon him, which interested me exceedingly. I told him to come back and see me on the following day, and I would show him something worth seeing, at the same time pro-

mising that nothing containing prussic acid should be in the room.

That afternoon I provided myself with six ounces of myrbane—a liquid which has precisely the same odour as prussic acid, but which like genuine maraschino, though smelling like prussic acid, contains none—and placed it out of sight in the same cupboard in which the prussic acid bottle had been. On the following day my friend returned; but no sooner had he crossed the threshold of my room than he rushed from it. As soon as he got outside of the door, and into the current of fresh air from the staircase, he stopped and began upbraiding me with having deceived him. I only laughed, and was just upon the point of telling him that his prussic acid theory was a mere 'figment of imagination,' when he spontaneously exclaimed, 'How funny! I don't feel ill to-day, and I am sure your room is as full of prussic acid as it was yesterday.' Here was a valuable piece of information. For it conclusively proved to me that the toxic properties of prussic acid did not exist in its odoriferous principles, but was only associated with them. To make a long story short, it ended in my friend—who, I think I may as well state, as there is no reason that I can see for his objecting to my giving his name, was Dr. Magrath, physician to the Teignmouth Hospital—putting his nose to the bottle without suffering the

slightest unpleasant sensation, while he could not tolerate my bringing the prussic acid bottle within yards of his olfactory organs.

These cases, then, show how the toxic effects of living germs and dead poisons, though associated with, do not necessarily exist in, the odoriferous principles of the deleterious agents.

This leads me to express an opinion regarding the origin and nature of the fœtor of disease, and I shall cut the matter short by at once saying that I do not think it proceeds from the tissues of the human body themselves, but wholly and directly from the disease-germs infesting it. In the same way, I believe that the odours as well as the colours of decaying animal and vegetable matter result from the emanations from and hues of putrefactive germs and nothing else. Indeed I believe that the fœtor of disease no more owes its origin to the human body itself than the fragrance of 'pot pourri' owes its to the vase. The human body, like the vase, being the mere container of the source of the odour. And this belief I found on the following facts.

A. Everything which destroys disease-germs or any other kinds of germs destroys the odours with which they are directly associated. Thus it is that not alone are sulphurous and carbolic acids, solutions of the sulphate and chloride of zinc and iron, &c., powerful deodorisers, but likewise powerful disinfec-

tants, from their destroying (I believe) the smell by killing the germs from which it emanates. Just as the odour of a flower vanishes (in great measure) when it ceases to live.

B. All ferment germs emit a peculiar odour during their active life as ferments. No matter whether they exert it in a cesspool, a dung-heap, a vat of wine, a barrel of beer, a lump of dough, a soup tureen, or a human stomach.

C. Most ferment and disease germs are highly odoriferous. Those producing the disease called wheat-bunt (the *Tilletia caries*) have so strong an odour that it is vernacularly called 'stinking smut,' and while sulphuric acid at once destroys its smell it at the same time destroys the germs' vitality.

D. The amount of odour evolved, as gauged by the olfactory nerves, may be said in every case to be proportionate to the quantity as well as the vital activity of the germs. Crace Calvert pointed out in the case of decomposing albumen ('Pharmaceutical Journal,' June 15, 1872) that the smell was in direct proportion to the development of germs. He says that in the case of albuminoid fermentation, the greater the number of vibrios produced, the stronger is the smell emitted from the decomposing matter.

E. In my papers on germs published in the 'Lancet' (June and July, 1881), I pointed out the fact which I had observed, that the dead bodies of

the dogs into whose veins I had injected the spores of a strongly smelling green fungus, had a strange fœtid odour, which, although not at all unlike the odour of putridity, could not possibly be due to any ordinary form of putrefaction, as they emitted it immediately after death, consequently long before any natural form of tissue decomposition could have time to manifest itself to the nasal organs. It was in fact, I believe, due to the ante-mortuary kind of fermentative putrefaction which disease-germs set up in the living tissues and blood of animals impregnated with them. In support of the correctness of this idea I may mention that in the case of the keeper of the snakes at the Zoological Gardens, killed in ninety minutes by the cobra's bite, his blood is described as having been found, after death, fluid, dark, and alkaline, and to have emitted a sickly 'sour' smell. In this case a smelling substance must have been manufactured in the man's body, as I have never been able to detect any sour smell proceeding from snake's venom, notwithstanding my familiarity with it. In other cases again, however, the germs which are introduced are odoriferous, as in the case of the dog just alluded to.

F. Different kinds of germs, like different kinds of flowers, possess not only different odours, but different colours ; and as nobody will deny that the green, yellow, brown, red, and black specks on cheese, stale bread, &c., owe their colours to fungi and fungi-

germ spores, I think I may venture to say that the green of the sarcina vomit, the black hue of mortification, and the greenish hue of human tissue decompositions are equally due to the colour of the germs present in each of these sets of cases. And further that the different odours emitted by patients labouring under different forms of disease are in like manner due to the differences in the species of the germs giving rise to them. Some have odours which we call sweet, others odours which we call nasty. The majority are certainly of the latter class, which is not to be wondered at, as the majority of disease-germs are fungi, and the whole fungus tribe may be said to possess more or less disagreeable odours. All toadstools have a more or less offensive smell, and I am not quite sure that if mushrooms were poisonous fungi, we should not even designate their odour as disagreeable. For agreeable and disagreeable odours are, like flavours, merely relative conventional terms for different kinds of mental impressions, what is regarded as an agreeable odour by one set of men being oftentimes designated as a disgusting stink by another. I need not, however, say anything more on this point, as I scarcely think any of my readers are at all likely to consider the effluvium from either the nose, mouth, skin, urine, or fæces of patients labouring under germ diseases peculiarly agreeable to their olfactory nerves.

It is thus then that I account not only for the existence of a special fœtor in a large class of diseases, but for there being a peculiar and specially characteristic form of odour emanating from patients labouring under different forms of disease. After having said this much, I must specially guard myself against being supposed to imagine that *all* the odours emanating from a human being either in health or disease are due to the presence of germs, for such an idea would be absurd. Many of the odours in disease, and almost all of them in health, are due to the presence of fragrant or stinking organic, though not organised, animal 'immediate principles.' The sweat, for example, often contains not only sebacic and butyric, but formic and valerianic acids. While in all cases of urinary suppression it is loaded with the ordinary urinary excrementitious matters, urea, uric acid, oxalate of lime, &c. &c., and acquires therefrom a distinctly urinous odour. Besides which many of the excretions eliminate in a state of almost chemical entirety the odoriferous constituents of the food. Even the breathing a putrid atmosphere from diseased bodies communicates to the excretions, both cutaneous, urinary, and fæcal, a disease-germ odour. This need not surprise us, however, when we remember that the urine and perspiration partake of the respired odours of vegetable matters very quickly,

and that it is a well-known fact that some students are seized with sickness and diarrhœa from working long in badly ventilated dissecting-rooms.

There is an interesting fact recorded by Dr. Stork which I ought not to omit mentioning. Namely, that he noticed that if he wore black clothes while dissecting typhus corpses, they always acquired sooner, and retained longer, the odour emitted from the bodies than light-coloured ones. From the preceding remarks it is seen that all the odours met with in disease are not to be supposed to arise solely from disease-germs.

But as additional evidence that I am correct in attributing the peculiar odours emitted from the bodies of patients labouring under germ diseases to the germs themselves, I would call particular attention to the fact that in all cases, without, I may venture to say, a single exception, there is a marked increase of the specific form of smell at the crisis, that is, at the time the germs are being eliminated *en masse*.

It is likewise observed that at what is called the crisis of a disease there is an unmistakable sudden outburst of activity in several of the secreting organs, the respective functions of which have, up to that time, been more or less in abeyance. For example, there are often sudden and profuse sweating, diuresis, and purging, just as if the great emunctories of the

body were no longer held in bondage, and, by the sudden removal of some pernicious controlling cause or another, were once more in a position to perform their normal function of eliminating noxious and effete matters from the system. This may be regarded as a highly probable idea, as it is invariably noticed that these sudden discharges from the skin, kidneys, and intestines are immediately followed by a decrease in the pulse and a fall of the temperature, coincident with a general improvement in the condition of the patient. The pyrexial and cerebral symptoms disappear, the appetite returns, the patient feels better, and convalescence all at once sets in. One of the most striking features in the case often being the sudden offensiveness of all the excretions, most notably of the perspiration.

The 'sweating crisis' was formerly a common expression in medical books, and no doubt it was an appropriate one. For we all know that sudden profuse perspiration is one of the most remarkable features in many forms of disease, particularly in ague and hectic, and is almost instantly followed by an amelioration in the symptoms. The perspiration smelling strongly. No doubt this arises from an elimination of the offending disease-germs having been effected by the sweating process. On more than one occasion I have particularly noticed that at the crisis of measles—that is to say, at the disappearance of

the eruption—there is not only occasionally a diarrhœa, but a distinct sweating stage, and that the perspiration is of a peculiar and sour smell. In fact most disease-germs, like almost every species of ferment germ, have a peculiarly sour odour. It is not alone the odour emitted by the sweat, however, that is stronger at that time, for the bad smells of the breath, the urine, and the fæces seem to be equally augmented. I was particularly struck with this in the case of a medical man, aged 45, whose case is sufficiently interesting to merit my giving a brief account of its most salient features, which are the following :—

On the third day after having breathed for a few minutes the disgusting effluvium from a freshly opened ancient cesspool, his friends noticed that his complexion was of a strange greenish-yellow hue, while he at the same time complained of feeling listless and uncomfortable. Shortly afterwards he was all at once seized with a violent rigor, which, after lasting for some time, left him so completely prostrated that he went to bed. On the following day he was in a state of high fever, and Dr. William Aitken diagnosed the case as one of acute blood poisoning from the cesspool gases. The case rapidly assumed a serious aspect, and from finding him one night, at half past eleven, in a state bordering on delirium, with a pulse of 124 and a temperature of

105°, Dr. Aitken not only regarded his case as hopeless, but left him in the expectation that he would die ere the morning. At twelve o'clock, however —that is to say, within half an hour after this— his skin suddenly burst into a profuse perspiration, which lasted for at least four hours, his night shirt and sheets being drenched with the sweat. Next morning the delirium was gone, the restlessness had ceased, the pulse had fallen to 108, his temperature to 101·7°, and, though fearfully weak and exhausted, he expressed himself as being comfortable. Then it was that I noticed the peculiarly strong increase in the fœtid odour of both perspiration and urine. On the morning following what we considered had been the sweating crisis, not only his perspiration, but his urine, literally stank. The urine at the same time was very high-coloured and loaded with a darkish-coloured lithate of soda deposit. Which fact, however, I chiefly attributed to its being concentrated, from having been passed in very small quantity on account of the profuse perspiration which had occurred during the night. He soon got well.

It has been noted by several observers that a peculiarly fœtid diarrhœa follows the crisis in cases of recovery from septicæmia. While I and others have been particularly struck with the stench of the first stools passed by patients after the crisis in erysipelas, puerperal, typhoid, typhus, and other

fevers. It is simply abominable, and even the smell of the urine is sickening to those who, like myself, possess acute olfactory nerves.

It may not be out of place for me here to mention that Gaspard and Cruveilhier long ago published the fact that they had noticed that the dogs which recovered after having putrid pus injected into their veins invariably passed terribly offensive black stools, as if the putrid poisonous matter was being eliminated by the bowels.

I think that I have now advanced sufficient data to justify my asserting that the fœtor met with in all cases of germ diseases—contagious jaundice, epidemic jaundice, acute atrophy of the liver, erysipelas, gangrene, phthisis, small-pox, malignant sore throat, &c.—is :—

1st. Not the direct product of the patient's diseased tissues and fluids, but the *normal odour* of the germs themselves infesting the patient's body and producing the disease under which he labours.

2nd. That the differences in the morbid odours of the breath, sweat, urine, and fæces in germ diseases are not dependent upon any differences in the composition of the patient's tissues and fluids, but entirely upon the differences in the species of the germs infesting them.

3rd. That the increase of the stench—which usually occurs immediately after the crisis—is directly

due to the increased elimination of the odoriferous germs.

Lastly. Just as my former colleague, Professor Williamson, one day told me, when I complained of the stink in his laboratory at University College, that he never objected to any smell which he was able to define by a chemical formula, so have I ceased to murmur against the stench of the sick-chambers of patients labouring under germ disease, since I discovered that it is not due to diseased filth—matter out of place—but to the normal odour of the healthy developing pathogenic germs which the patient's excretions are most laudably doing their best to get out of their involuntary suffering host's—the patient's —body. And further, that it is my, as it is every other practitioner's duty, not alone to avoid thwarting their endeavours, but to aid by every possible means in our power this eliminating process, even though by so doing we may in some instances necessarily increase instead of diminish—at least for a time—the intensity of the offending smell.

The next group of liver diseases about to be considered is an important one, as it includes all the incurable as well as remediable forms of hepatic affections which possess the power of inducing

Jaundice from Obstruction.

Examples of jaundice from obstruction are not only frequently met with, but are due to a multitude of widely-differing morbid anatomical conditions. In some cases the diseased state giving rise to it originates in the hepatic tissue itself; as, for instance, when cancerous tumours press upon and obstruct the ducts, or inspissated bile blocks up their canals. In many the obstructing cause originates in the gall-bladder, as when gall-stones induce it. In other cases it is the common bile-duct alone which is at fault; while in yet others the source of the mischief lies beyond both the liver and its appendages. As, for example, when a duodenal ulcer's cicatrix, or the pressure of a tumour of the head of the pancreas, occludes the intestinal orifice of the bile-duct. But what has been hitherto considered the strangest part of all is, that while in the majority of cases the gall-bladder is found enormously enlarged and distended with bile, in others it is met with not only empty but shrivelled up. This being the case, one cannot feel surprised that no single form of affection in the whole range of medical nosology has proved so puzzling in its minute details as the pathology of jaundice from obstruction. And yet notwithstanding all the erroneous suggestions regarding it which have so long appeared in our printed books, when the reflected light of modern physiological and pathological science is focussed upon

it, it appears to be almost as simple of comprehension as A B C.

I shall now endeavour to show that this is really the case. But as the unravelling of the more complicated parts of the mechanism of the different varieties of jaundice as the result of obstruction, in order to be clearly understood, requires not only the adjunct of attention and reflection on the part of the reader, but a categorical as well as a lucid exposition by the writer, I shall begin by offering a few general remarks, equally applicable to the merely transient and the most permanent forms of jaundice from obstruction, which may serve as a sort of preliminary clue to the solution of the mystery in which it has been hitherto imagined to be involved. And in order to make the remarks terse as well as lucid, I shall tabulate them in the form of hepatic pathological axioms, which, even should they prove devoid of any other merit, will at least possess the salient advantage of enabling me to put forward my views on the matter in a few plain words, and thereby considerably simplify the comprehension of this hitherto considered most abstruse part of the study of the pathology of complicated hepatic disease.

The axioms regarding the mechanism of jaundice from obstruction which I desire to put forward are :—

1st. That jaundice never arises from the congenital absence, or from the accidental destruction, of a human gall-bladder, for the simple reason that a bile reservoir

is in no case absolutely essential to animal life. For just as in the horse, the deer, the rat, and other animals that possess no gall-bladders, the biliary function is perfectly well carried on, so also it may be in the human subject, labouring under a congenital or an accidental deficiency of the gall-bladder. In such cases, if, as in the animals above referred to, the hepatic ducts are pervious, the secreted bile finds no difficulty in reaching the intestines. In the 'Edinburgh Medical Journal' (May 1861, p. 1045), Dr. Alexander Simpson reports a case of this kind occurring in a child, who died when only a few weeks old, in whom there was no trace of the existence of a gall-bladder. On laying open the duodenum, the orifice of the bile-duct was seen in its ordinary situation, and a drop of pale bile was expressed from it. On tracing the duct back into the liver, it was found to pass up undivided into the horizontal fissure, where it at once divided and sent branches into the hepatic tissue of the right and left lobes.

2nd. Jaundice is an inevitable concomitant of complete occlusion of a normally formed common bile-duct in any part of its course from its beginning to its end.

3rd. Jaundice may, and frequently does, arise from occlusion of the hepatic duct itself.

4th. Jaundice cannot possibly, in any case whatever, arise from an obstruction of the cystic duct, be

the obstruction ever so complete or ever so permanent.

5th. Death never did, nor ever can, arise from occlusion of the cystic duct *per se*; for just as a gall-bladder is not a necessity to human life, neither is the presence of a cystic duct in the least degree essential.

6th. When the common bile-duct is obstructed, the gall-bladder and bile-ducts, if normally formed, are invariably found after death DISTENDED with dark, thick, tarry bile.

7th. Whereas, when the hepatic duct is obstructed, the gall-bladder after death is always found to be EMPTY, and frequently even shrivelled up.

8th. While, when the cystic duct is obstructed, the gall-bladder after death is found neither *empty* nor *distended with bile*, but filled with a *white glairy secretion* possessing no analogy whatever to bile, either in appearance, physical properties, or chemical composition, being in fact nothing more or less than the pent-up *normal* mucous secretion of the mucous membrane of the gall-bladder.

For the logical explanation of the mechanism of this condition, which has hitherto led the uninitiated to imagine that the function of the gall-bladder had in such cases undergone a marvellous change, see my remarks at p. 1083, where I consider the diseases special to the gall-bladder.

9th. In explanation of the mechanism of the mode of emptying and shrivelling up of the gall-bladder, when the hepatic (instead of the cystic) duct is obstructed, I may mention that even in many cases, where the patient has succumbed from the direct effects of the jaundiced condition itself, the gall-bladder has been found after death not only to contain *not a trace of bile*, but to be perfectly empty and shrivelled up to the size of a writing quill. This at first sight strange phenomenon I account for in the following wise. While no bile can find its way through the obstructed hepatic duct into the gall-bladder to fill it, all the gall-bladder's own normally secreted mucus (from the cystic and common ducts being still pervious) readily finds its way out of it into the intestines, and consequently no accumulation of pent-up glairy white mucus takes place in the organ, as occurs when the cystic duct is obstructed.

While again from the gall-bladder's natural function of becoming distended and acting as a reservoir for the excess of bile secreted during the intervals of digestion, being in abeyance, the organ is never called upon to dilate at all, and as a natural consequence, from the want of use, it gets smaller and smaller, until it at length dwindles down to the diameter of a mere tube.

When the common bile-duct is obstructed, not

only is there in general severe jaundice, but also a gall-bladder distended with bile—concentrated bile from the fact that although the bile is prevented from finding its way out of the gall-bladder into the intestines, its secretion still goes on, and more and more of it is forced into the gall-bladder, until the viscus gets enormously distended and can dilate no more—then, from a process of capillary osmosis being set up, the aqueous and more soluble parts of the bile are absorbed by the fine capillaries of the walls of the viscus, and gradually and slowly its contents become more and more concentrated, till at length, if the patient only survives long enough, the gall-bladder is found after death to be chokefull of a thick, viscid, black, tarry-looking bile. This is the true and simple explanation of the hitherto supposed to be obscure facts, that a gall-bladder may be found in one case of jaundice as big as a child's head, and full of bile. In another, empty and shrivelled up to the size of a writing quill. While in a third case it may be found fully distended, and yet containing no bile whatever, but, in its place, a glairy white mucous secretion, about the supposed obscure pathology of which one occasionally sees such roundabout theories ventilated in medical journals.

For further details on these points see the chapters specially devoted to diseases of the gall-bladder.

11th. In no case of jaundice resulting from an

obstruction to the outflow of the bile is the obstruction ever found either in the secreting cells of the liver itself or in the gall-bladder, but only in the bile-ducts—either before or after their exit from the hepatic tissue.

12th. In jaundice from obstruction, by far the most frequent seats of the obstructions met with are in the ductus communis choledochus.

13th. All cases of jaundice arising from an impediment to the flow of bile along the ducts of the liver originate in one of the following three perfectly distinct pathological conditions :—

a. It may arise from an accidental obstruction in the course of the ducts, as from gall-stones, hydatids, or other foreign bodies.

b. From a congenital deficiency of the bile-ducts.

c. From closure of the outlet of the common duct. For example, from the cicatrisation of a duodenal ulceration, from the pressure of hepatic tumours, of the distended transverse colon, from organic disease of the head of the pancreas, or other growths of the neighbouring organs.

14th. The main feature in the pathology of jaundice from obstruction lies in the fact, that although no impediment whatever exists (at least in the first instance) to the normal secretion of the bile by the hepatic cells, yet, from the natural passage of the

secretion into the intestines being arrested, it gradually accumulates behind the point of obstruction. Is re-absorbed by a process of capillary osmosis into the general circulation. The serum of the blood becomes surcharged with the bile-pigment, allowing it to transude through the coats of the cutaneous capillaries into the rete mucosum of the cutis vera and stain it yellow, thereby producing the tint of the skin which we designate jaundice.

15th. While at the same time, from no bile finding its way by the natural channel into the intestines, the fæces are of a pipeclay colour instead of being stained of the usual brownish tint by the bile-pigment.

CHAPTER XI.

BILIARY CONCRETIONS.

Introduction and General Remarks on Inspissated Bile and Calculi.

JAUNDICE arising from a stoppage to the flow of bile through the gall-ducts by a solid biliary substance of some kind or another is, in my opinion, a very much more common occurrence in this country than appears to be generally imagined. And this, I believe, arises from the fact that the clinical history, pathological significance, and chemical constitution of all kinds of solid biliary substances are not only very imperfectly understood, but their symptomatology as given in books is in many respects actually erroneous. For example, not only do we find solid biliary substances like gall-stones and inspissated bile lumped together as if they were one and the same pathological product, but there is abundant evidence in both our home and foreign medical journals to show that many writers on hepatic affections are but very imperfectly informed regarding the manner in which either of these substances produces jaundice, and are unaware that inspissated bile as well as a

gall-stone is capable of producing fatal occlusion not only of the bile-ducts, but of the intestines, abscess of the liver, and even malignant disease of both the liver and gall-bladder. I shall, therefore, endeavour to throw all the light of modern science that I possibly can on this as yet but very imperfectly appreciated subject. In order to be able to do this successfully, it will be necessary to begin at the very foundation, and clearly demonstrate what is the real difference between the chemical composition and physical properties of the various forms of biliary concretions. For it appears to me that much of the ambiguity springs from the confused notions that still exist regarding the true nature of the solid biliary substances which obstruct the flow of bile through the gall-ducts into the intestines.

Physical Properties of Biliary Concretions.

Concretions of inspissated bile and true gall-stones appear to be lumped together in the minds of most persons as if they were merely different shaped and sized, identically constituted substances, merely called by different names. Hence, in the conversations and writings of medical men, one not only occasionally, but frequently, finds these two terms employed synonymously. A gross and, in a clinical as well as a pathological point of view, most important error. For while the one is the direct result of a

truly abnormal pathological formative process, in as far as quantity is concerned, the other is merely due to the relative proportions of the solid and aqueous ingredients of the otherwise normally secreted bile having become accidentally disturbed from extraneous causes. In fact, the two kinds of solid substances which I designate as inspissated bile and biliary calculi, bear no resemblance to each other whatever, either in chemical composition, physical properties, or pathological origin. Except in so far as they are both products of the biliary secretion.

In order to avoid the risk of being considered either unjust or guilty of exaggeration, I shall quote a reported case which abundantly proves that even among the most enlightened of our medical brethren, some speak of all biliary concretions as if they had not only a common origin, but a similar chemical composition. For example, Dr. Quain has published the case of a Greenwich pensioner, aged 91, from whose gall-bladder, after death, he says he removed '735 stones,' while, from the description given of the substances removed, it is clearly seen that they were not 'stones,' but merely hard masses of inspissated normal bile. For they are described as being friable, of a dark greenish colour, and possessing rough irregular surfaces. A combination of properties which, even without any chemical analysis, stamps them in the mind of the initiated as not being

gall-stones at all, but merely agglutinated masses of inspissated bile, consequently not pathologically speaking morbid products, in the true sense of the word. Only agglutinations of normally secreted physiological materials.

I have cited this case in order to show how, if an otherwise medically-learned writer, such as Dr. Quain, falls into an error as regards the nature of biliary concretions, we cannot be surprised at the undeniable fact that our whole British and foreign medical literature teems with precisely similar mistakes.

As it is quite as much in the interests of clinical medicine as of pathological science that such errors should be as speedily as possible consigned to oblivion, I shall now proceed to explain what is the difference between a gall-stone and a concretion of inspissated bile.

A gall-stone may be as big as a goose's egg, or so small as to be invisible to the naked eye. Notwithstanding which it invariably possesses a definite composition if not even also a definite structure. Whereas concretions of inspissated bile are never large, and, no matter what their size, shape, or position may be, are invariably mere heterogeneous structureless aggregations of ordinary biliary materials. Again, while the external surface of a gall-stone may be rough, mammillated, or even spiculated like a hedge-

hog, or as smooth and polished as the surface of a mirror, it never presents the rough irregular jagged indescribable formation of surface invariably presented by merely agglutinated bile. Every gall-stone has a describable form. No matter whether it be globular, oval, elongated, cylindrical, and truncated; of a triangular, hexagonal, or polygonal shape; or even of the extraordinary form of branched coral (a representation of a specimen of which is given in Plate V. of Cruveilhier's 'Pathological Anatomy').

When dry a gall-stone is hard and unyielding to the pressure of the finger and thumb, even when it is of a steatomatous nature. Whereas, when dry, a concretion of inspissated bile is brittle, and crumbles into gritty dust under the pressure of the fingers. I must here further allude to a very peculiar and rare form of steatomatous gall-stone, or its future description might lead to misconception, from the fact that the term 'stone' is usually associated in the mind with the property of hardness; and although not generally known, there are such things as perfectly soft biliary concretions. At least when first passed and warm, they are both soft and pultaceous—so soft that they can be kneaded into any shape by the fingers, like a piece of putty—and, even when old and dry, are no harder than a piece of stearin, and consequently can easily be scratched with the nail or cut with a knife, just as a stearin candle

can, and that, too, for precisely similar reasons. Namely, that they are of a fatty nature. Indeed, I believe them to be nothing else than masses of cholesterin in its preliminary stage of crystalline formation. Biliary products of this kind are rare; but, as will be subsequently seen, both I and others have met with them assuming the appearance of dirty-white barleycorns, orange-pips, grapes, and pigeons' eggs, leading even the most experienced to commit awkward mistakes.

As regards colour again, while masses of inspissated bile, be their size or shape what it will, are invariably dark in colour, generally blackish-green, though occasionally brownish-red, gall-stones, on the other hand, are of a great variety of colours, sometimes as white as newly fallen snow, sometimes as black as ink. They may be yellow like beeswax, green, brown, or even red. In my collection I have an extraordinary coloured one, which I took from the gall-bladder of a woman who died in 1862 with bronzed skin. It is of the size of a small hazelnut, and has a brown outside shell-like crust, with a blue slate-coloured kernel-like interior. Specimens of this kind, I fancy, must be rare. For I have never seen another like it, notwithstanding that I have seen most of the biliary concretions in nearly all of the great pathological museums of Europe.

Some gall-stones are entirely composed of pure

white crystalline cholesterin. I possess one the size of a pigeon's egg of this kind. It is slightly nodulated externally, crystalline internally, and of an alabaster colour.

A more curious fact still is that some gall-stones actually resemble (not only in form and size, but in general appearance) 'pearls of the purest water.' I have in my collection six beautiful white small pearl-like gall-stones, which were found in the gall-bladder of a Danish ox. So like are they to pearls of the purest water, that they would deceive the eye of even an experienced judge, if he were not allowed to handle them. I have also a beautiful specimen of a brownish-red, distinctly stratified biliary calculus, taken from the gall-bladder of a Danish woman. Both the pearls and the red calculus were presented to me by Professor Panum on my visit to Copenhagen in 1874. Being duplicates of specimens existing in his museum.

There are several beautiful differently coloured and shaped specimens of gall-stones in the Museum of the Royal College of Surgeons. Some of them like yellow beeswax. Some like tailor's white chalk. Some pinkish in colour. Some perfectly globular in shape. Some columnar. Some truncated cylinders. One specimen of the latter shape, over an inch in length and three-quarters of an inch in diameter, has a small globular stone set into one of its ends; the two to-

gether forming a good specimen of a ball-and-socket joint. On section, or better still on fracture, gall-stones present either a crystalline, a homogeneous, or a stratified appearance, whereas concretions of inspissated bile are always totally structureless.

Sometimes, though very rarely, gall-stones have foreign materials as nuclei—a blood-clot, a shrivelled-up entozoon, or, it may be even, a fragment of inspissated bile. While, as far as I am aware, concretions of inspissated bile never possess nuclei.

FIG. 12.

Section of gall-stone showing internal crystalline structure.

Specific Gravity of Biliary Concretions.

All biliary calculi—true gall-stones—very greatly diminish in weight after being exposed for a few days to the air, from their sometimes containing, when freshly passed, as much as 50 per cent. of hydroscopic water. It would therefore, I think, be decidedly to the interests of medical statistics if gentlemen would refrain from recording the weights of calculi before they are dried. For when newly passed and moist, their weight is of no clinical importance whatever, since it is often quite as much due to the quantity of water they accidentally contain as to the solid substances of which they are composed. This remark is not uncalled for, seeing that a great deal of nonsense has been written regarding the specific gravity of freshly passed gall-

stones, which nonsense has given rise to an important error in the mode of procedure recommended for their detection. I made some particular experiments on this subject, which were as follows :—

On putting thirty gall-stones taken promiscuously from my collection, and therefore representing all kinds of comparative shapes and sizes, and which, from their having been put up as dry specimens, had become air-dried, into a vase of distilled water, I found that about one half floated and the other half sank to the bottom of the jar. On putting seven freshly passed stones—that is to say, stones which had been passed within forty-eight hours—into the same jar of distilled water, six sank to the bottom, and only one, a small one, the size of a pea, with a whitish, smooth, soapy feeling and tailor's-chalk-like surface, floated. Having thus satisfied myself that while the vast majority of freshly passed stones are heavier, only one-half of dried stones are lighter than water, I proceeded to take the specific gravity of those that were heavier than water, and I found that it varied very greatly, from 1000·1 to 1025. The heaviest stone being of the last-mentioned specific gravity. It had a slightly nodulated mulberry-looking surface, was of the colour of a dirty-white piece of alabaster, and the size and shape of a wood pigeon's egg. On making a section of it three-quarters through with a saw, and then splitting the remaining uncut portion,

in order to show its appearance both on section and fracture, I found it consisted entirely of beautifully white, almost chemically pure, large crystals of cholesterin, densely packed together ; the crystalline appearance being in this, as in all other cases, best seen on the fractured surface. From this series of carefully conducted observations it may now, I think, be confidently affirmed that, contrary to previous teachings, *almost all* gall-stones, when fresh, are heavier than water, varying in specific gravity from 1000·1 to 1025, but when air-dried many of them, by losing moisture, become lighter even than distilled water, and hence float upon it. Those that float on water are in general found on section to have a hollow centre, from their unsolidified substance while drying shrinking from the centre towards the hard external shell, just as many a molten mass of silica, by hardening on its surface during the cooling process more quickly than in its interior, presents on section a hollow centre. Concretions of inspissated bile, on the other hand, no matter how dry they become, are never hollow in the centre, but uniformly heterogeneous throughout, and always heavier than water.

I may here incidentally allude to a fact of some importance, as it may save others from falling into error regarding the exact nature of calculi passed from the human bowels, which is, that while I was busy in 1859, preparing a series of intestinal con-

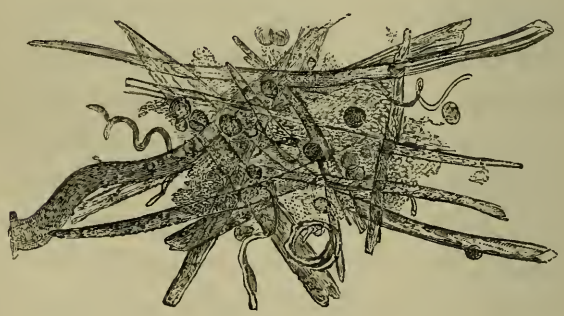
cretions for exhibition along with their analyses at the Pathological Society (see vol. xi. p. 86 of the 'Transactions'), Professor Sharpey, who always took great interest in pathological as well as physiological analyses, while sitting talking to me in my laboratory at University College, all at once exclaimed, 'Oh! I have a much rounder gall-stone than any of these passed by the rectum. I shall let you see it.' Going immediately to his private room on the same landing, the door of which was nearly opposite mine, he returned with a perfectly globular concretion in his hand; it measured exactly one inch and a quarter in diameter, and had a hard smooth exterior of a dark greenish-black colour, just like a dark gall-stone. Being in the act of preparing to wash my hands when he gave it to me, I popped it into the clean water in the basin, when to my surprise it floated like a cork, being far more buoyant than any gall-stone under similar circumstances would have been. Never before having seen a gall-stone either so light or so beautifully globular, I turned to Professor Sharpey, and said, 'Well, if that is really a gall-stone, it is a curiosity. Will you let me cut it? For I don't think it is a biliary calculus at all.' 'Oh yes,' replied Professor Sharpey, 'you may do what you like with it, for it is of no use to me.' In a few minutes it was divided in two, and the felt-like appearance it presented on section told me at once that it was not a

gall-stone at all ! but an intestinal oat-hair concretion, exactly the same, except in colour (being somewhat darker), as some I had just been examining from Mr. Liston's collection in the college museum.

When teased out with needles, the dense felt-like substance of these calculi is found to consist of the hairs of oat-seeds, mixed with intestinal mucus and inorganic salts ; their ashes being composed of lime and magnesia, with a little soda. Those in Liston's collection are about an inch in diameter, and twenty in number ; they were all passed at different times by a patient whose chief food had for many years consisted of oatmeal porridge.

The subjoined woodcut, taken from the Morbid Anatomy part of my 'Histological Demonstrations,'[1]

FIG. 13.



Oat-hair Intestinal Calculus.

represents the matters composing these calculi when examined under the microscope.

[1] 2nd Edition, p. 192. Longman & Co.

How to detect Biliary Concretions voided by Stool.

Very great difficulty is experienced not alone in the detection of small masses of inspissated bile, but even of large gall-stones that have been passed along with the fæces. This in great measure arises from the false advice given for their detection in books. All of which say that biliary concretions are to be detected by adding water to the stools, and that if gall-stones are present they will be found floating on the surface. A more ridiculous piece of advice it is scarcely possible to conceive ; for, as already shown, the vast majority of freshly passed gall-stones are heavier than water, and even some of the air-dried ones may have a specific gravity actually reaching to 1025. So that in almost no case whatever, when freshly passed, could they be found floating on water. I have never yet been able to detect a gall-stone in this way. The plan I recommend is, to mix the stool freely with water, and either decant the supernatant fluid, and then add fresh portions of water till the whole of the soluble matter is removed, or better still to strain the mixture through a hair-sieve. The gall-stone in either case remains behind, and can be readily detected.

Moreover, it is always necessary to explain to the searcher that gall-stones may be as small as pins' heads, or as large as hen's eggs. As black as the

fæces themselves, or as pale and white as frosted silver or snow. As irregular in shape as a triangle, or as round as a marble. As smooth and as white as polished ivory, or as rough and as dark as a mulberry.

On one occasion I got from a lady's maid a gall-stone the size of a very large field pea, which she actually found the first time she strained the stool through a sieve, although she had been fruitlessly searching for gall-stones for months on the old plan of adding water to the stool, in the expectation of finding the gall-stones floating on the surface. How many gall-stones this maid had missed before she adopted my plan of search, I know not; but thirteen were found in the stools within two months after its adoption. Twelve of which specimens I exhibited to the Pathological Society.

Not one, but a dozen, nay, even a whole hundred gall-stones, as large as ordinary garden peas, have been found in one single motion of the bowels. While, in a remarkable case which occurred while the patient was taking the waters of Carlsbad nearly three hundred, varying from the size of millet-seed to that of a pea, are said to have been discharged with one action of the bowels. In a case of this kind I should suppose that the stones did not come through the channel of the common duct from the gall-bladder, but were all at once

passed into the intestines through a direct ulcerated opening between the gall-bladder and intestines.

In looking for gall-stones in the stools, great care must be taken not to confound the seeds of fruits passed by the bowels with gall-stones, and *vice versâ*. A mistake which often happens. As was the case with a specimen I exhibited in connection with a series of gall-stones to the Pathological Society, which had not only deceived the patient, but actually deceived many of the medical gentlemen who saw it at the meeting. Until I demonstrated its true nature by cutting it in halves. An exactly opposite kind of mistake is equally liable to be made. Namely, that of mistaking true biliary concretions for seeds.

In proof of how exceedingly easily this is done, and in order to show how very difficult it is sometimes to avoid making mistakes of this kind, and missing gall-stones even when they are actually before our eyes, I shall relate what happened to myself. When, notwithstanding my twenty years' experience of gall-stones of all shapes, colours, sizes, and consistency, I fell into the error of taking two steatomatous biliary concretions for lemon-seeds! As a warning to others, I think I had better tell the tale in full. It happened thus :—

In September 1881, Mr. John Gay, surgeon to the Great Northern Hospital, sent me a lady. Who had been for some time under the care of a clever

general practitioner, aided in consultation by one of our leading London (titled) consultants, without (as she expressed it) their doing her any good. What their treatment may have been I know not; but I assuredly know that their diagnosis was correct. For a clearer case of gall-stones it has scarcely ever been my lot to see. There was paroxysmal pain and tenderness over the liver, bilious urine, pipeclay-coloured stools, sickness, and jaundice. Lasting for a few days. Followed by a total subsidence of all signs and symptoms, and, after ten days or so of repose, bursting out again afresh. Again to subside, and reappear after a longer or shorter interval. On no occasion—at least while I saw her—was the interval sufficiently long to admit of the patient's being said to be convalescent. My opinion was that she had a lot of small gall-stones, or a quantity of inspissated bile, in her gall-bladder. Which came away in small quantities at a time, temporarily blocked up the common bile-duct, and was then voided by stool. But against this supposition stood the salient fact that the stools were carefully searched for the offending substances, without avail. The longer I live, however, and the more experienced I become with the difficulties besetting the path of gall-stone stool detection, less and less importance do I attach to the negative data of 'not finding a stone,' and I think the reader, after perusing what

I am now about to say, will feel strongly inclined to endorse this opinion. The lady above alluded to was under my care from September 1 till December 17, that is to say, fifteen weeks. During which period she had four separate attacks, and, as I saw her many times, I had ample opportunities afforded me of modifying, correcting, or entirely changing my opinion of the nature of her case, had new facts and signs presented themselves. But the more I saw of her, the more convinced was I that the diagnosis of repeated discharges of small biliary concretions being the cause of her jaundice was essentially correct, and fortunately for my own personal information, during the few last times I saw her, from the appearing of 'pure bile' in the vomit crucial evidence was at length afforded not only of the exactitude of the diagnosis, but of the success of the treatment.

At the termination of one of her attacks I had the benefit of Sir William Gull's opinion, and on another occasion when she had signs of ileo-cæcal valve irritation, no doubt from what is a common occurrence, the lodgment of a concretion in the valve—which the patient, however, thought was a return of an inflammation of her uterine appendages, from which she had at one time suffered—I had the additional advantage of her former surgical attendant, Mr. Spencer Wells's, advice. Both of these gentlemen, I need scarcely say, came to the same conclusion

regarding the nature of the case as I had done. In fact, no other opinion could under the circumstances possibly be arrived at; yet, as there was never a gall-stone found, the patient was not only dissatisfied with the treatment, but an unbeliever in the diagnosis. Now comes the episode of my being completely deceived by mistaking two biliary steatomatous concretions for the seeds of a lemon. At the termination of the second last of the attacks for which I attended her, and while the stools were yet of a creamy hue, and consequently without any distinct evidence of bile in them, I saw two pale turtle-dove coloured oblong masses in the soft fæces; they looked *exactly like lemon seeds*, and knowing that she was then in the daily habit of drinking the juice of squeezed lemons, and receiving no contradiction from the professional nurse, who was holding the chamber-pot in her hand, I concluded that a couple of lemon-pips had been accidentally thrown into the utensil, and so I thought nothing more about them. What, however, was my dismay when at my next visit I learned that *no* lemon-seeds could by any possibility have got into the pot! From the juice only of the lemon, carefully freed from seeds and pulp, being ever brought into the sick-chamber. Now to me all was clear; the supposed seeds were oblong soft steatomatous concretions, moulded into the shape of lemon-seeds during their passage through the bile-

ducts ; their appearance, size, and soft nature completely accounting for the pain being so slight, as well as for their having hitherto invariably escaped detection. Consequently I gave orders that whenever any more of these seed-looking substances appeared, they should be kept for me to examine. An order which was disregarded. For on the next occasion of their appearance, the patient examined them herself ; tested their nature by squeezing them, and, from finding them soft and pultaceous, her medically untutored mind 'knew that they had nothing whatever to do with the jaundice'—at least so she positively affirmed—and, without more ado, she consigned them to the w.c. Of course I knew that she was mistaken about the nature of the soft substances ; but how to convince her of that fact was the difficulty. For I had on more than one occasion observed, while endeavouring to explain to her the nature of her case, she appeared to be one of those persons who, while sceptical of the diagnostic knowledge of the doctor, entertain an implicit belief in the infallibility of their own notions of the etiology of their symptoms. Consequently, after unsuccessfully trying the effect of a little mild reasoning as to the steatomatous nature of the bile substances she had passed, I thought it better to leave her in the happy delusion of the correctness of her opinion. In fact, no good could possibly have arisen from my contradicting her. As

it did not matter what her peculiar views on the subject were, as time would of itself prove her to be in the wrong. Which idea was much sooner verified than I expected. By her vomiting a very few days afterwards a large quantity of pure bile, thus affording positive proof that the obstructing biliary concretions had actually been passed, and that the bile-ducts between the gall-bladder and the duodenum were once more perfectly free. For otherwise pure bile could not have found its way into, and been vomited from, the stomach; at least not unless there had previously been bile-duct or gall-bladder perforation, to induce an opening into the stomach through which the bile could have directly passed from the gall-bladder in spite of the obstruction. After this evacuation of bile I saw nothing more of the case, so I cannot say whether or no all the biliary concretions that were originally in the gall-bladder had then been got rid of. One thing, however, I do know, and that is that owing to the treatment by biliary solvents, administered to her in the shape of soda and taraxacum and alkaline salicylates, she was, when I last saw her, on the high road to complete recovery.

After what has just been said the reader will not be surprised to learn that 'sad experience,' the most infallible of all guides to human success, may be said to whisper to us that the only possible way to avoid

failures in searching the excretions for voided biliary concretions of all kinds is to pick out from them everything, though it be as small as a pin's head or as big as a hen's egg, which differs in appearance from the general mass. No matter what its colour, form, or consistence may be. For biliary concretions may, as we have seen, vary from the whiteness of newly-fallen snow to the blackness of ink; be round as a ball, oblong like an orange-pip, or as flat as a small fragment of note-paper. Even inspissated bile is sometimes passed by stool as tiny dark thin scales, the true nature of which is only discoverable on their being treated with strong sulphuric acid, which instantly turns them of a bright crimson hue. While again, as regards consistence, the concretions may be as hard as stones, or as soft as newly melted wax. My advice is therefore to pick out everything that at all differs in appearance from the general constitution of the stool. Wash, dry, and test it, physically, chemically, and microscopically, if need be; for then, and then alone, is it possible to avoid mistakes. But even in that case a negative result is not necessarily to be considered proof positive that no biliary concretion has been passed by stool; far less is it a proof that an offending concretion has not escaped from the hepatic appendages into the intestines.

My reason for so saying simply arises from my knowledge of the fact that a gall-stone, after being

passed out of the bile-ducts, may quietly remain in the intestines for days, weeks, months, or even years, and ultimately pass out along with the fæces when not expected, and consequently at a time when it is not being looked for. As a curious example of this, I may mention that a gentleman, some time after having ceased to have symptoms of gall-stone, and when he had come to the belief that the diagnosis of his case had been wrong, as no biliary concretions were found in his stools, heard, while he was sitting on the closet, something hard fall into the pan. On looking at it, he saw a dirty-white-looking thing among the fæces as big, he said, as a pigeon's egg; but never dreaming that a gall-stone could be anything like that, he drew up the plug, let it slip away, and thought nothing more about it, until I began one day explaining to him the many curious appearances gall-stones assume. When, from my description, he at once knew that the egg-shaped dirty-white-looking mass which he had voided was the gall-stone he had so long fruitlessly searched for.

A still stranger anecdote is the following. Last year a lady whom I was attending for gall-stones one day showed me a substance (as big as half a walnut when cut transversely), of a polished whity-yellow look, with irregular facets upon it. Which she said was kept in her family as an heir-loom. It being supposed to be a piece of the back-

bone(!) of an aunt of hers. Which she had passed at stool. The history of it was this. Long after the patient had ceased to have hepatic symptoms, one day she passed by stool a hard substance which hurt her considerably, and, on looking at what had come away, she saw, as she thought, a lump of bone in the fæces. She took it out. Washed it, showed it to her family, who unitedly diagnosed it as 'a piece of her backbone' which had come away, and henceforth carefully preserved it as a family heirloom. I saw that it was merely a cholesterin concretion, and had the cruelty to dispel the interesting family delusion.

I might cite many cases of a somewhat similar character to the above; but I think that these two examples are sufficient to show how even patients who have been in the habit of searching for gall-stones in their stools may make mistakes when they are actually passed, merely from their coming away not only at a period when they are not being looked for, but at a time long after all the symptoms of the biliary derangement have vanished. Which fact is of course not in the least surprising, when it is known that gall-stones, after being passed into the intestines, may not only quietly remain there for years before being voided by stool; but may, as will be subsequently shown, never be voided at all, and their existence only be discovered at the *post-mortem*.

Prognosis of the Number of Gall-stones in any given Case.

In the earlier part of my career as a teacher at University College Hospital, I taught, as I had myself been taught, that the number of facets found on any voided gall-stone was a trustworthy indication of the probable number of stones in the patient. While if there were no facets upon the stone found in the stools, it would in all probability be the case of a solitary calculus, and the patient might be safely congratulated that he would have no more attacks. Within the last few years I have learned that this is utterly false; for although the presence of two, three, four, or more facets tells plainly that other stones have been formed in the body along with the one that has been voided, the absence of facets is no proof whatever that the stone has been a solitary one. Three stones are now in my possession, as big as small hazel-nuts, and, although four such came away within a few days of one another, not one has a vestige of a facet upon it. The details of this case will be given under the heading of gall-stones being mistaken for cancer.

The Chemistry of Biliary Concretions.

It will now be shown how the different kinds of biliary concretions, which I respectively designate inspissated bile and gall-stones, bear quite as little con-

stitutional similarity to each other in a chemical as in a physical point of view. For while those of the gall-stone class are found to consist chiefly of cholesterin, with mere traces of bile-pigment, concretions of inspissated bile are composed of biliverdin and other organic and inorganic bile products, with scarcely any cholesterin entering into their composition.

Gall-stones again are not even so much as formed in the same way as concretions of inspissated bile, for like urinary calculi they are formed by gradually repeated depositions of pathological products, in so far as quantity is concerned ; whereas, as will be immediately shown, concretions of inspissated bile are mere heterogeneous agglutinated masses of normal biliary solids, resulting from a deficiency of their normal aqueous solvent. Hence, as will presently be seen, gall-stones, that is to say true biliary calculi, and concretions of inspissated bile possess but one feature in common—namely, being the direct products of the biliary secretion.

Chemical Composition of Inspissated Bile.

The following is the result of an analysis which I made of 120 grains of hard, irregularly shaped green masses of inspissated bile of the average size of small barleycorns, taken promiscuously from my collection :—

Water	5·4
Solids	94·6

The solids consisted per cent. of

Bile pigment	84·2
Cholesterin	0·6
Inorganic salts (iron, potash, and soda)	15·2

On comparing the above analysis of inspissated biliary concretions with the analysis of specimens of healthy bile taken from human gall-bladders given at p. 83, it is seen that they actually consist of nothing beyond the normal solids of the biliary secretion, being in fact merely normal bile deprived of its aqueous elements.

Chemical Composition of Gall-stones.

On chemical analysis I found that twelve air-dried gall-stones, taken promiscuously from my collection, and of an average weight of 31 grains each, yielded

Water	4·2
Solids	95·8
	100·0

The solids consisted per cent. of

Cholesterin	98·25
Pigment and mucus	0·50
Inorganic salts	1·25

In 1856 I exhibited to the Pathological Society a gall-stone which had ulcerated its way out of the gall-bladder, and become lodged in an artificially formed cul-de-sac of the intestine.[1] The mass consisted of three jointed pieces of nearly equal size, collectively being of a pear shape, as if they were a cast of the gall-bladder, and weighing 450 grains. On analysis I found that they consisted of

Water	2·543
Solids	97·451
	100·000

The solids consisted of

Cholesterin	90·346
Mucus	2·218
Colouring matter and biliary resin	4·242
Inorganic salts	0·661

The difference in the proportion of the ingredients in the small and in the large calculi is perhaps not altogether due to an original difference in composition, but partly to the fact that while the above analysed small calculi came direct from the gall-bladder, the big one had sojourned, and been subjected to the chemical action of the digestive juices for many years perhaps, in the intestinal canal.

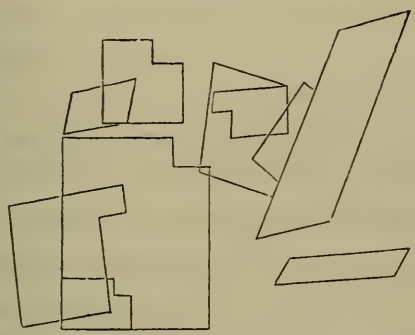
[1] This case is fully reported in the *Pathological Society's Transactions*, vol. viii. p. 235.

In 1858 Dr. Gibb brought to the physiological laboratory at University College a curious specimen of a calculus which he found encysted in the gall-bladder of a man aged over 70. The patient had been a hard drinker, and his liver was both chronically atrophied and nodulated. The gall-bladder, which was quite embedded in the hepatic tissue, contained four calculi. Three were free, and a fourth, the largest, weighing 66 grains, was encysted close to the gall-bladder orifice of the duct. Which it completely plugged up. Not a single drop of bile, Dr. Gibb said, was found in the gall-bladder. On chemical examination it was found that all the stones consisted of more or less pure cholesterin. The purest of the four being the one which weighed 66 grains. It had the further peculiarity of its external surface consisting of a loose foliaceous layer of crystals. Which, when examined under the microscope, looked so like crystals of sulphate of soda, that I mistook them for such, until strong sulphuric acid revealed that they were cholesterin, by producing with them the beautiful play of colours, presently to be alluded to, characteristic of that substance.

Before proceeding further with the subject of biliary concretions, I will redeem the promise made in the physiological chapter of saying a few words more on the nature and chemical properties of cholesterin.

Cholesterin, although a biliary product, appears to be a normal constituent of healthy human blood, from which it can readily be extracted by treating dried serum either with boiling alcohol or ether. From the extract made with the former, the cholesterin crystallises in the flat rhombic plates shown in fig. 14. From the latter in fine needle-shaped crys-

FIG. 14.



Cholesterin crystals in the form of fine transparent four-sided plates of various sizes and shapes. The crystals are freely soluble in hot alcohol, from which they are re-deposited on cooling.

tals. Pure cholesterin is insoluble in water, dilute acids, and alkaline solutions.

With strong sulphuric acid it gives a beautiful play of prismatic colours, which are best seen when the reagent is applied to it on a clean white porcelain capsule.

When a solution of iodine is added after applying the strong sulphuric acid, a still more beautiful play of blue, green, yellow, and red colours is obtained.

Cholesterin occasionally appears in the urine in some hepatic diseases where a large quantity is being formed, and, for some as yet unknown reason, crystals of it are frequently to be met with in serous exudations. Such as pleuritic, peritoneal, ovarian, and hydrocele fluids.

The foregoing details are not to be regarded in the light of merely curious physical and chemical data, but as important pathological facts. Which every practitioner ought to be fully acquainted with, and bear in mind when he is called upon to treat a doubtful case of gall-stone, or a suspicious one of inspissated bile. For, as I have just shown, while in the former case he has an actual chemical vice in the system to correct by his therapeutic agents, in the latter case he has only to attempt to retain the normal bile in its usual state of aqueous fluidity. Two entirely different matters, requiring the aid of different therapeutical agents. For, although what is called a gall-stone may occasionally, but only rarely, contain a large amount of biliary pigment mixed up with cholesterin, that which is properly known under the title of inspissated bile never contains a sufficient amount of cholesterin in it to be recognisable, except by chemical analysis.

Such being the physical and chemical differences existing between the two great classes of biliary concretions—inspissated bile and gall-stones—in

future when I speak of concretions of inspissated bile, I wish it to be distinctly understood that I never, by any chance whatever, mean a gall-stone, no matter however small in size it may be, but solely and simply an irregular structureless agglutination or concretion of concentrated, thickened, hardened bile. Of variable size, shape, and colour, but of only slightly variable composition. While on the other hand, when I speak of a gall-stone or a biliary calculus, I always mean a true stratified or crystalline stone. Always containing a maximum of cholesterin, and a minimum of bile-pigment. Varying, it may be, in size from a naked-eye invisible point to that of a goose's egg or bigger, and in colour from a pure pearly or snowy whiteness to a light or dark brownish red or black hue. According to the greater or less admixture of colouring matter it accidentally contains.

It is necessary for me here to call atention to a fact of some clinical importance. Namely, that gall-stones and inspissated biliary concretions may co-exist not only in the same patient, but in precisely the same locality at one and the same time. This I have several times noticed ; but a much more striking case than any I have met with is one recorded by Mr. Wale Hicks of a woman aged 60, who died from atrophy of the liver induced by obstruction of the common bile-duct by a large gall-stone. The descrip-

tion given is, that 'the gall-bladder was small, and contained a little thin bile ; the cystic duct and part of the common bile-duct were occupied by a large gall-stone, which also projected into the gall-bladder. The hepatic duct and its branches were very much enlarged, and filled with dark green masses of nearly solid inspissated bile.'

Inspissated Biliary Affections.

One occasionally hears medical men speaking in an offhand manner of cases of jaundice that they imagine they have met with as the result of a blocking up of some one or other of the bile-ducts by concretions of inspissated bile, as if they were things of almost everyday occurrence. Whereas from over twenty years' personal experience I know that while cases of jaundice from impacted gall-stones are exceedingly common, those from inspissated bile are exceedingly rare. Consequently I opine that inspissated bile is a much-maligned substance, being frequently blamed for mischief it never produces by a class who talk as if many hepatic affections, both with and without the concomitant of jaundice, arose from it. While others again, running to the opposite extreme, not only ignore the existence of inspissated bile as a pathological product of any importance whatever, but occasionally go so far astray as to confound its signs and symptoms with those arising from malignant disease. An error

which, as will be presently shown, has not always only been revealed at the *post-mortem* examination, but even, in some cases, during the actual lifetime of the patient by its being voided by the stools, and the morbid supposed cancerous symptoms immediately vanishing, it may be never to return.

Etiology of Inspissated Bile.

Before a concretion of inspissated bile can form, either in a gall-bladder or in a bile-duct, there must have existed a preliminary, though it may have been a merely temporary transitional stage of thickening of the bile. Which stage I shall now allude to, and no better published example of it can I find than that given by Dr. Hunter in his book on 'Diseases of the Army in Jamaica,' in which he describes a typical case. Which is briefly as follows:—

'A few days before the death of a patient from pulmonary consumption jaundice came on, and at the autopsy there were found signs of superficial inflammation all over the liver, the lower surface being bound to the stomach by adhesions. The gall-bladder was full of bile. The common duct was filled with a thick ropy black molasses-like bile coagulum. So firmly implanted in the duct was this thickened bile that pressure on the gall-bladder failed either to dislodge it, or to force a single drop of the thin bile contained in the gall-bladder past the coagulum into

the duodenum. It was even not until after the introduction of a blowpipe into the duct, and by considerable force, that air could be driven along it into the gall-bladder. The duct was subsequently laid open from the gall-bladder to the duodenum, and not a trace of any other obstructing cause beyond the thick ropy tarry bile coagulum could be found.'

This is such an excellent example of the antecedent and transitional stage between healthy fluid and the formation of hard concretions of inspissated bile from a mere deficiency of water, that I need adduce no further examples of it.

Symptoms of Inspissated Bile.

The symptoms which characterise an obstructive attack of hardened inspissated bile are in general, like those of gall-stones, sudden in their onset and painful in their duration. They usually begin during the period of fasting, and are therefore most common between ten at night and ten in the morning. Sometimes they are little more than a dull aching feeling in the right hypochondrium. At others they amount to acute paroxysmal colic, sometimes increased by direct pressure, yet frequently relieved by soft manual frictions. When the attacks are moderate there is neither jaundice nor vomiting; when they are severe there are usually both. The attacks generally last from

twelve hours to twelve days. Then in general subside as suddenly as they came on. As a rule, the patients are what may be called bilious subjects. Many of them come of a bilious family ; and usually they can recall to mind a previous attack during which they were either actually jaundiced or very nearly so. If the attack be not sufficiently severe to produce discoloration of the skin, it seldom gives rise to much gastric derangement, nothing perhaps beyond a little flatulency and constipation.

Now as regards the pathology of this state of things, the explanation is very simple. A viscid state of the bile gives rise to the formation of small concretions of hardened biliary matter ; these plug up and irritate the small ducts sufficiently to produce pain. If only a few of the small ducts are plugged up, there is not a sufficient amount of biliary disturbance to produce jaundice. If, on the other hand, the concretions are sufficiently large or numerous to block up some of the large bile-ducts, especially the hepatic or the common bile-duct, jaundice is the inevitable consequence. When small concretions cause pain, they only do so as long as they remain in the smaller ducts. For as soon as they reach the larger ducts, which they are not big enough to plug up and irritate, all pain instantly ceases, and the patient feels suddenly as well as ever he was. If the fragments of the hardened bile, so soon as they reach the larger duct,

are floated away, as they frequently are, into the intestines, they mix with, and are expelled along with, the fæces, their symptoms vanish entirely, and nothing more is ever heard or seen of them. This, however, only happens under exceptionally favourable circumstances. For sometimes the patient is not only a victim to paroxysms of pain at irregular recurring intervals from a succession of concretions forming in the ducts; but occasionally the concretions become so large that they block up the ducts permanently, and either a fatal jaundice or the formation of fatal abscesses in the parenchyma of the liver behind the seat of the obstruction is the result.

As a striking illustration of the theory that jaundice may arise from inspissated bile blocking up the 'small ducts' of the liver, and not only give rise to all the symptoms of gall-stone, but even end fatally, I shall cite the case related by Dr. Grainger Stewart at p. 627, vol. xviii. of the 'Edinburgh Monthly Medical Journal.' The case was that of a man aged 24, who died jaundiced with symptoms of impacted gall-stone, and at whose necropsy it was found that the right lobe of the liver was enlarged, the left atrophied, and throughout the whole organ were numerous abscesses, varying from the size of a pea to that of a walnut, while the ramifications of the bile-ducts in the substance of the liver itself were so dilated that many of them admitted the point of the finger. Most of them

contained 'partially inspissated bile and a finely granular blackish material; but some contained pus in addition, others almost pure pus.' When this history is scientifically interpreted, as there was no impacted gall-stone found, and the gall-bladder and cystic duct were of normal size, while the ductus communis choledochus was also perfectly normal, it is impossible to doubt the fact that the enormous dilatation of the ducts, as well as the jaundice and the death of the patient, were entirely due to the obstruction caused to the flow of bile through the small ducts by their being blocked up with inspissated bile.

To Dr. Handfield Jones we are indebted for the publication of a case ('Pathological Transactions,' vol. v. page 150) of 'universal jaundice,' where on *post-mortem* examination the cause was found to be the plugging up of the outlet of the common bile-duct by a quantity of fine 'sandy matter' consisting entirely of bile-pigment.

The presence of inspissated bile in the ducts has even proved fatal to infants. A well-marked case of this kind is reported in the 'Northern Journal,' under the head of fatal jaundice in a new-born child. Where on *post-mortem* examination the cause of death was found to be occlusion of the common gall-duct by 'an indurated cord-shaped plug' of inspissated bile.[1]

[1] *Northern Journal of Medicine*, vol. i. p. 240.

Occasionally, inspissated bile blocks up and irritates the small bile tubes in the parenchyma of the liver, producing no symptoms whatever beyond pain. These cases are exceedingly difficult of diagnosis, from its being only by adopting a system of eliminating the diseases which cannot exist, and by a process of scientific reasoning from analogy, that one can by any possibility arrive at a correct diagnosis. As a good illustration of this, I may relate the result of a consultation which I had with Dr. F. Weber in February 1880. The patient was a leading London banker, of about 60 years of age, of whom I received from Dr. Weber the following history.

For two years past he had been at irregular intervals, of from one to two months' duration, seized with severe, sudden, spasmodic attacks of pain in the right hypochondrium, which, after lasting from twelve hours to a day or two, as suddenly ceased as they began ; the patient feeling instantly as if quite well. The pain Dr. Weber said he could compare to nothing else than 'tic,' and morphia was the only thing that relieved it. During the time of the attacks all the bodily functions seemed perfectly normal. For although the patient was the subject of an habitually weak digestion, he had neither vomiting nor sickness, constipated nor loose bowels. The urine was natural in every respect, and there was neither jaundice nor sallowness of the complexion. In former years he

had suffered from oxaluria; but that had passed away, and there was no reason whatever to suspect the existence of a renal calculus. In fact, as Dr. Weber said, if it were not for the 'unaccountable symptoms of the paroxysmal abdominal tic,' the gentleman ailed nothing. My familiarity with the oftentimes anomalous and puzzling symptoms arising from biliary concretions led me to suggest the possibility of inspissated bile being the cause of the paroxysmal pain. So the patient was without more ado brought into the room, and I proceeded to examine him.

He was an average-sized man, of spare build, and, though not robust, had a healthy look. The account he gave of himself tallied in every particular with the history Dr. Weber had given. The seat of pain was directly to the right of the navel, and occupied an area bounded above by a line drawn transversely parallel to the lower margin of the xiphoid cartilage, and below by another across the abdomen at the level of the umbilicus. On percussion the liver was found to be perfectly natural as regards size, and not the least tender on firm pressure. He at the time of examination was quite free from all pain, but he assured me that even when he had an attack, rubbing the painful part gave relief. As nothing tangible could be discovered in the abdomen to account for the paroxysms of pain coming on at irregular intervals, I felt more convinced than ever that the case was one

of inspissated bile. So I cross-questioned the patient very minutely as to his previous personal and family history, and learned that he was not only a member of 'a liver family,' but that he had once had an attack of jaundice—though it was forty and more years ago. In fact, when he was only a boy of twelve years of age. Here now was a direct clue to the cause of the paroxysmal pain, and my next question was 'At what time do the attacks usually come on?' The reply was, 'Before breakfast.' This was another small, though not unimportant, link in the diagnostic chain. Fasting, as I before said, being the usual time when the spasms from inspissated bile come on. This history was sufficient for me. No longer had I the slightest doubt as to the nature of the case, and on communicating to Dr. Weber my ideas of its rationale, he at once put the not uncalled-for question, 'But how can such small particles of inspissated bile, by plugging up the small ducts of a non-sensitive organ like the liver, cause such severe pain, while they yet produce no jaundice?' To this I replied that although the liver was a non-sensitive organ, the bile-ducts when irritated by the presence of foreign bodies—as, for instance, when a gall-stone blocks up one of them—give rise not only to pain, but to most excruciating agony, which ceases as suddenly as it begins, so soon as the offending foreign body finds its way into the intestines. The relative amount of pain caused by small fragments of inspis-

sated bile blocking up the smaller being proportionally the same as that produced by large calculi blocking up the bigger ducts. The amount of pain induced in any case being directly proportionate to the bulk of the foreign body in comparison with the calibre of the duct in which it happens to be impacted. I further pointed out how the absence of jaundice in many of the cases of inspissated bile is readily explicable, not alone on account of merely one, or at most only a few, of the small ducts being blocked up, but on account of the free anastomosis of the ducts with each other preventing in general any local accumulations of bile sufficiently large to produce jaundice, as well as from the fact of the hepatic and common ducts remaining pervious. Whereas the sudden disappearance of the symptoms, and the patient's immediately afterwards feeling perfectly well, were equally readily accounted for by comparing the case with one of paroxysmal pain from gall-stones, to the suddenness of the disappearance of which it offers a most striking analogy, both as regards cause and effect. Dr. Weber having expressed himself as satisfied with these pathological explanations, the case was prescribed for on rational, in contradistinction to empirical principles. That is to say, the *cause* of the disease, not its symptoms, was attacked ; soda and taraxacum were accordingly given, in order to induce a larger secretion and flow of thin fluid, instead of a

small one of thick viscid bile. The result of the treatment may be judged of by the following extract from a letter regarding another matter which I received from the patient's wife on September 6, 1880. In it she says :—

'My husband remained quite well until three weeks ago [that is to say, six months from the time I saw him : G. H.], when he had a return of the pain. Five weeks after leaving off the medicine.'

The Symptoms produced by Inspissated Bile are oftentimes mistaken for those of Cancer.

At first sight one would think it almost impossible for a well-educated medical man to mistake the symptoms produced by such an apparently insignificant thing as inspissated bile for those caused by such a grave morbid product as cancer. Nevertheless, not only is such often the case, but the reason of it is very simple. Both seldom produce much jaundice, diffuse hepatic pain, or considerable constitutional disturbance. I shall cite two typical illustrative cases of this kind, and the first I shall select is a fatal one, about which I was consulted by letter in 1864 by Mr. Cripps, of Cirencester. The case was diagnosed as one of jaundice arising from cancer of the liver, and treated accordingly; but at the *post-mortem* the jaundice was discovered to have been merely due to the presence and accumulation of inspissated bile in the ducts. Mr. Cripps kindly sent

me the gall-bladder, with the ducts attached to it, for examination, and I found impacted in the common duct a quantity of hard granular-looking black particles of inspissated bile. While the gall-bladder itself contained only one globular mass, the largest of all, it being almost of the size of a small pea. Which on examination was found to be merely made up of a number of small granular particles, similar to those in the common duct, feebly agglutinated together, but forming a mass too large and too firm to pass through the vesical orifice of the cystic duct, as the loose particles had no doubt done. These specimens of inspissated bile, when taken collectively, I consider form one of the most instructive in my collection, from their showing what a very trifling quantity of misplaced biliary matter may cause the death of a patient ; for even when the whole were put together they did not weigh twenty grains.

I shall now relate the history of a most instructive, and certainly to me one of the most interesting hepatic cases I ever met with. The case is that of a Northumberland squire whom I saw in May 1877, along with the present President of the Royal College of Physicians. He was sent up to London to consult us conjointly by Dr. William Murray, physician to the Newcastle Infirmary ; and as the case was not only a puzzling one, but presented many interesting peculiarities, I shall give it somewhat fully.

The patient, a strongly built tall man of 59 years of age, from labouring, like some others, under the erroneous belief that he would get more information out of his two consultants if he saw them separately, came to me and got my opinion, without even so much as alluding to Sir William Jenner's name. Then he went by himself to Sir William Jenner, and got his opinion, but did not tell him either what my opinion was, or even so much as that he had ever heard of my existence. Now after having done so, however, to his great annoyance he found himself in an awkward position. For the two consultants had expressed entirely different opinions, both of the nature of his case and its treatment. In fact, not only did the opinions he got disagree in mere details, but they were absolutely opposed to each other in every single particular. For while Sir William Jenner had told him that his case was one of organic enlargement of the liver, and given him at the same time to understand that, being such, it was incurable—thereby confirming the opinion of one of the medical men he had previously consulted in the North—I had told him his disease was merely a slight, temporary, and curable one. That all the bad symptoms, even that of a state of collapse from which he said he had suffered, were entirely due to the accidental blocking up of the hepatic ducts by inspissated bile. What was he now to do? Return home with these con-

tradictory diagnoses ? Try a third doctor ? Or what ? He knew he had disobeyed Dr. Murray's instructions ; for he had not even so much as delivered to either Sir William Jenner or myself the letters of introduction he had with him. After some little, no doubt unpleasant, reflection, he made up his mind to call on one of us, make a clean breast of his folly, and ask what he had better do under the circumstances.

On the following day he called and communicated to me his dilemma. So I at once wrote to Sir William Jenner and asked for a consultation. Well knowing, as I told the patient, that if he had described his case to Sir William Jenner in the same way as he had done to me, there could not have been any material difference in our opinions. For equally trained minds must of necessity draw the same conclusions from the same data, when placed before them in the same light. The consultation was held, and the chief facts elicited were :—

That the patient complained of a dull aching pain and a distinct tenderness on pressure over the whole of the hepatic region. The dull area of which was considerably increased. There was no nodulation to be detected. The patient had an unhealthy sallow look, but no true jaundiced or cachectic tint. The conjunctivæ were more of a pale dirty green than of a yellow hue. He looked depressed and anxious, but not particularly ill. He said that he had most

discomfort and pain at the pit of the stomach, and, when asked to point to the spot, he placed his finger on the end of the xiphoid cartilage. He said his hepatic symptoms had begun six months before (in December 1876), when he was seized with a strange feeling of malaise, sickness, pain in the stomach with tenderness on pressure, accompanied by fruitless efforts at vomiting. Thinking that it was a severe attack of indigestion, he put on a poultice, and took a dose of castor-oil. The result was magical. In a few hours he felt perfectly well. The whole attack did not last, he said, above six hours. He remained well for fourteen days, when again suddenly the same symptoms reappeared. The same line of treatment, however, had no effect whatever. Rapidly he grew worse, until he became so bad that he fell into a state of complete collapse. In which state he remained for twelve hours. There was no jaundice. By the end of four days he quite recovered, and remained perfectly well for four months. When he was again seized while asleep in bed. At twelve o'clock at night he suddenly awoke with pain at the stomach and intense sickness, so intense that he vomited freely, though only once. For a couple of hours after vomiting he felt as if a cord was bound disagreeably tight round his waist. Exactly on a level with the xiphoid cartilage. At the same time there was a general uneasiness and great tenderness over the whole of the

hepatic region. This last attack happened just three weeks before he came up to London. During the three weeks which intervened he had felt very poorly and good for nothing. The feeling of the cord, the uneasiness, and the tenderness in the liver had been continuous. With more or less slight daily exacerbations.

After talking the case carefully over together, and discussing the *pros* and *cons* of every one of the anomalous symptoms, as well as taking into consideration the fact that there was but a slight and incomplete obstruction to the flow of bile into the duodenum, there being no distinct jaundice, Sir William yielded so far to my view of the case that he allowed me to prescribe for the patient. I strongly suspect, however, that he doubted my diagnosis. For before leaving he somewhat significantly remarked that if my prognosis of what he termed 'the minute pathological diagnosis' should chance to be confirmed by the recovery of the patient, the diagnosis itself never possibly could. Strange to say, however, by one of those unforeseen lucky turns in the wheel of fortune, not only has the prognosis been verified by the patient's complete recovery, but the diagnosis itself was confirmed, even to its minutest details, within four days after the consultation, in the following most unexpected wise.

Believing, as I before said, that all the symptoms

arose from inspissated bile blocking up the ducts, I prescribed for the patient a strong alkaline cholagogic cathartic mixture, with the view of flushing them with liquid bile, and thereby washing out of them the inspissated hardened masses of the secretion. What was the result? I shall leave the subjoined letter, which I received from the patient himself on the morning of the fourth day after the consultation, to say. I give it *verbatim*, simply omitting names and the last paragraph, which has no reference to the point under consideration.

'On my way to Newcastle on Saturday I had two premonitory attacks—the first from 8 to 9.30, and the last from 12.30 to 2.30.

'I slept in Newcastle, and had another and very severe attack, commencing at 8 and continuing until 7. The pain and sickness were dreadful. After the violence of the attack had abated I had a motion, in which were found the substances I now forward for your examination, and shall be glad to know if, in your opinion, they are what are called gall-stones.

'I had an excellent night's rest, sleeping from 11 till 6.30; and to-day, notwithstanding the blue pill I took last night, feel myself perfectly well.'

On reading this letter I felt doubtful as to what the hard masses might possibly turn out to be. For I could not for a moment imagine that the 'minute pathological diagnosis,' about which I had been twit-

ted, was so soon to be verified. However, on applying to one of the masses the proper chemical tests, it instantly yielded a result which incontestably proved it to be a mass of inspissated bile.

Having satisfied myself that the small dark masses forwarded were undoubtedly inspissated bile, I wrote in reply that he might tell the doctor that he could easily convince himself that the diagnosis had been confirmed by simply pouring a few drops of strong sulphuric acid over one of the masses placed on a white plate, when the acid would at once turn the green biliverdin into a magnificent scarlet colour, thereby proving conclusively that the mass was composed of inspissated bile. Fortunately the treatment of the case has proved as successful as the diagnosis was correct ; for although six years have elapsed since it was begun, the patient is not only still alive, but in the enjoyment of robust health. Never once having had a return of the attack.

Death may occur from Inspissated Bile blocking up the Intestinal Canal,

after having safely escaped from the gall-bladder, by direct ulceration of its coats ; and that, too, when neither doctor nor patient has even so much as suspected its existence. A most instructive case of this kind was brought by Dr. Pye Smith before the Pathological Society in 1854. The case was that of a woman aged 69, who, after a fortnight of slight pain

in the right hypochondrium, began to feel sick and vomit bile. A few days later she brought up about a gallon and a quarter of bilious fluid, after which the bowels became constipated. On the fifth day the vomited matters, though less in quantity, had a most offensive smell, and therefore an intestinal obstruction was suspected. She died on the sixth day. On the *post-mortem* examination the jejunum was found completely obstructed by a solid oblong mass four and a half inches long, two and a quarter inches in circumference, consisting of hardened inspissated bile of a dark colour and pungent odour. 'On raising the liver, its under surface was found occupied by firm fibrinous adhesions surrounding Glisson's capsule, in the midst of and protected by which was an ulcerated communication from the almost absorbed gall-bladder into the duodenum, half an inch below the opening of the common bile-duct. It is inferred that through this the above-described large mass of inspissated bile had passed.' (Pathological Society's 'Transactions,' vol. v. p. 163.)

Treatment of Inspissated Bile.

The treatment of the active symptoms which arise from a blocking up of the bile-ducts by concretions of inspissated bile is in general simple enough, and may be summed up in a few words.

During the attack apply hot poultices to the seat of pain. Give the patient a hot bath. Administer

an anodyne along with belladonna, to dilate the ducts, and follow it up with a smart purgative.

Prophylactic Treatment.—In order to prevent the formation of concretions of inspissated bile, it is necessary to administer to the patient every now and again those chemical substances which are most effective in keeping the bile fluid. Soda given in the shape of bicarbonate or sulphate is transformed in the system into the glycocholate and taurocholate of soda, the two active and normal biliary solvents, and so long as they exist in the bile in sufficient quantity there is no chance of a concretion of inspissated bile forming. This theory having a clinical as well as an experimental basis to rest upon, my usual plan of preventive treatment is either to give one or two drachms of sulphate of soda in a bitter infusion every morning before breakfast, or from twenty to thirty grains of bicarbonate of soda along with a drachm of taraxacum juice in a bitter infusion every night at bedtime at regulated intervals, for a month or so, according to the constitution of the patient and the severity of the symptoms.

Further details of treatment will be gleaned from the special chapter on treatment, and what has yet to be said on the preventive treatment of gall-stones.

Gall-stone Affections.

The clinical history of biliary calculi forms, as a whole, one of the most interesting as well as important

chapters of hepatic affections. For not only, as before said, are gall-stones the commonest of all the causes which produce jaundice, in this country at least, but their symptoms are frequently mistaken for those arising from entirely different forms of disease—renal calculi, cancer of the liver, &c. Thereby not only leading to an erroneous prognosis, but even to fatal errors in treatment. Added to which few are aware of the great number and variety of collateral fatal forms of disease to which gall-stones give rise. For example, they occasionally, as will be presently shown, induce abscess as well as cancer of the liver, perforation of the stomach and intestines, enteritis and peritonitis, hæmorrhages, &c. It will therefore be necessary for me to treat the subject of gall-stone affections very fully, and the more so as I have a great many new facts to adduce.

The Etiology of Gall-stones.

Of the etiology of gall-stones it may be truly said that as yet little definite is known. But as we are already in possession of some very important data connected with their clinical history and chemical composition, I think I am in a position to adduce some more facts in connection with their pathological formation beyond what I have already given while treating of biliary concretions in the aggregate. Gall-stones, and indeed biliary concretions of all kinds, are, like urinary calculi, very frequently hereditary.

Not once, but several times, I have had more than one member of a family under my care, suffering from either concretions of inspissated bile or actual gall-stones. While writing this (1879) I have a lady aged 42, and her son aged 18, under treatment for gall-stones, and the mother of this lady was at one time similarly affected. So here is an example of the hereditary descent of the disease in at least three generations. As there are several cases already recorded, where gall-stones have appeared in two successive generations, I may pass on to the next point, namely, that Prout, Budd, Trousseau, and many others, have called special attention to the frequency with which biliary and urinary calculi are found in the same individuals, especially in those of a gouty constitution. Scudamore in 1823, and several other older writers, have even described a condition of chronically congested liver in gouty patients, to which they accorded the special name of 'gouty hepatitis.'

Gall-stones are much more frequently met with among women than among men. The proportion generally given being as three to two ; but if I may form an opinion of the proportion occurring in Britain from observations made in my own and my friends' practices, I should say that as many as about two women are affected with the complaint to every one man. This I attribute to their usually more obese

habit of body, from natural constitution as well as from their less active modes of life.

The fact that gall-stones are more common among women than men is, however, rather extraordinary, seeing that they are oftentimes associated with the gouty diathesis, it being a well-known fact that gout is a much more common complaint among the male than among the female sex. A striking example of this exists in my own family, where, notwithstanding that for four consecutive generations gout has affected almost every male, not a single female member of the family is known to have suffered from it, although even their male descendants have had that disease, and in some instances in a severe form.

Gall-stones may occur at any period of life between the cradle and the coffin. Indeed at the post-mortem of a child not a month old, was found a gall-stone. And what is more curious still is the fact that gall-stones may actually form in a child's body while it is yet in its mother's womb. This we know to be the fact from Bouisson having found three gall-stones in the gall-bladder of a newly born infant with an impervious common bile-duct. This can astonish no one, however, who has paid attention to what I previously said about the secretion of bile beginning as soon as the fœtal liver is formed, which is about the end of the third month of intra-uterine life, as from that moment onwards the formation of gall-stones becomes of course possible.

Gall-stones occur in varying proportions at different periods of life. From a comparison of the statistics which have been given by different European writers, I think it may be said that in every 1,000 cases

750 occur in persons of over 40 years of age.
200 „ „ between 30 and 40
40 „ „ „ 20 and 30
10 only „ under 20

This fact as regards age strongly supports my theory that the kind of food used, as well as the bodily activity, has greatly to do with the formation of gall-stones in predisposed constitutions. For while in early life, when the vital processes are in greatest activity, the hydrocarbons of our food—the elements out of which gall-stones are formed—are rapidly and completely consumed, in later life, when the vital processes are less active, only a portion of them is used up by the frame, and the excess, which is neither eliminated from nor consumed by the body, is generally deposited and stored up in and around its organs and tissues as fat. While in the predisposed a portion of the fat-forming principles of the food is transformed by the liver into the crystalline fat called cholesterin, and is deposited in an insoluble condition from the bile in the shape of gall-stones. In connection with the important part played by the

hydrocarbon elements of food in the formation of gall-stones, I may here mention that, by a strange coincidence, two gentlemen who came to me on the same day labouring under gall-stones, confessed that they were exceedingly fond of, and in the daily habit of eating, salt bacon. While the one said he had constantly had it for breakfast during the previous nineteen months, the other declared he had scarcely for a single day breakfasted without partaking of it for very nearly as many years. The accidental circumstance of these two gall-stone sufferers having made to me the confession of their fat-bacon proclivities, on one and the same day, naturally produced a profound impression on my mind—so profound indeed, that ever since I always ask gall-stone patients if they are addicted to bacon-eating, and it is really surprising how many of them say that they are not alone fond of bacon, but of all other kinds of fatty, oleaginous, and starchy foods. Be it remembered that a purely starchy or saccharine diet is nearly as favourable to fatty formations and cholesterin deposits as a diet of strictly oleaginous materials, from starch being converted into sugar, and sugar into fat, in the animal economy. Consequently all animals freely supplied with starch or sugar become fat, and seeing that cholesterin, which is the main ingredient of gall-stones, is a true crystalline fatty substance, it is easy to understand how a copious

supply of farinaceous foods, in the predisposed, tends to the development of biliary concretions. This is not a mere theory, but a demonstrable fact ; for Dr. Crisp has ascertained that sheep fed and fattened with sugar, as well as stall-fed oxen, who consume a large proportion of starchy foods, are particularly liable to gall-stones.

From among the facts I gleaned while on a tour in Russia in 1874, I may mention that I noticed, in the Museums of both Moscow and St. Petersburg, that not alone were gall-stones exceedingly numerous, but many of the specimens were likewise of very large size. Indeed, the largest gall-stone I have ever seen is one in the Pathological Museum of the Civil Hospital at Moscow. It is nearly as large as a goose's egg.

The cause of the Russian gall-stones being of such unusually large dimensions is probably partly due to the habit of the natives of consuming large quantities of oleaginous foods. Not butter ; for that is not only an unheard-of luxury among the people, but actually an unknown product of the dairy. The Russian peasantry not even so much as knowing how to make it ; fat and suet being used in its place. There may, however, exist another cause, one originating in the medical profession itself. For throughout the whole of Russia, except in the large towns, it is at a very low ebb, and the absence of proper treatment in liver disorders no doubt favours the

formation of gall-stones. While the same absence of treatment, later on, favours their attaining to a large size. From gall-stones being relatively more common in cold than in warm latitudes, and bilious disorders thought to be more general at the fall and the spring of the leaf, a cold and damp atmosphere has been supposed to favour their development. After having given considerable attention to this point I have, however, arrived at the conclusion that it is neither the cold nor the damp of the northern latitudes which directly interferes with the biliary functions, but the kind of food which the damp cold necessitates. It is a thoroughly understood axiom in experimental physiology that, in order to sustain the weight of the animal body at a low temperature, more oleaginous and fat-producing foods are required than are necessary for the purpose in a warm atmosphere.

We all know that while Englishmen living in the cold climate of the Arctic regions tolerate, if they do not even actually enjoy, a meal of oily blubber, they instinctively turn from such food with disgust when living at the equator. And, as before said, fatty and fat-generating foods are conducive to the formation of gall-stones, from the mere fact, if from none other, that cholesterin, their chief component, is a crystalline fat. This theory, that it is the food more than the cold which accounts for the frequency of gall-stones among the natives of the northern hemisphere, is

further borne out by the observation that in Norway (which is certainly both as damp and as cold as, if not indeed much colder than, the southern parts of Finland, Sweden, and Denmark) I found proportionally fewer gall-stones in the museums of Christiania and Bergen than in those of Helsingfors, Stockholm, Gothenburg, and Copenhagen. Which I account for by the fact of the inhabitants of Southern Norway employing more of a fishy and less of a fatty kind of food. I may further mention, as an additional fact in favour of my theory regarding the etiology of gall-stones, that lardaceous livers sometimes contain a large amount of cholesterin. So much so that, by the spontaneous evaporation of an ethereal extract of the hepatic tissue, crystals of it are deposited in abundance. As having an important bearing on their etiology, I may here further remark that I was particularly struck with the fact that the majority of the gall-stones I saw in Russia and Finland were white. A precisely similar circumstance, though in a less striking degree, I had previously noticed in connection with those exhibited in the museums of Sweden. Where gall-stones seem to be almost as plentiful as in Russia. While in the Pathological Museum of Christiania, I particularly noticed that instead of the general colour of the stones being white, as in Russia, Sweden, and Finland, it was decidedly the reverse. Indeed, I saw among the

Norwegian gall-stones four perfectly black nut-sized ones, said to have been removed from one and the same human gall-bladder, and a tumbler full of equally black small (pea-sized) ones, which were said in like manner to have been all removed from another gall-bladder at the *post-mortem* examination of a patient who was not until then known or even suspected to have gall-stones. The mere fact of the colour of the gall-stones in Norway being different from those of Russia is of itself evidence that while in the latter country the fatty elements greatly predominated, in the former the pigmentary entered largely into their constitution. As is the case with inspissated biliary concretions, whose etiology is quite different. I may further incidentally remark, in connection with what I have already said regarding the constitutional tendency to form urinary and biliary calculi co-existing, that in Norway urinary calculi seemed to be almost as rare as gall-stones (probably from the purity of the drinking water), while both forms of calculi appeared to be about equally abundant in Russia. It is worthy of remark, in connection with the etiology of gall-stones, that any derangement of the biliary function likely to induce the introduction of the bile acids into the general circulation may possibly favour their formation. For Feltz and Ritter observed that crystals of cholesterin (the substance of which the vast majority of gall-stones are composed) appear in

the serum of the blood of animals into whose circulation bile acids have been artificially introduced ('Comptes Rendus,' April 12, 1875). The number of gall-stones which may form in the body is unlimited. Morgagni tells us of 3,645 having been found in one gall-bladder, and Dr. Otto put up a specimen of a gall-bladder that contained no less than 7,802 ! Of course, the more numerous they are, *cæteris paribus*, the smaller is their relative size.

Just as it occasionally happens that two or more urinary calculi, after attaining a considerable size, become glued together, and surrounded by a common covering of a different kind of urinary deposit from that of which the calculi themselves are composed, so I noticed in the Anatomical Museum of the University of Moscow that there was a rare and beautiful specimen, in which two gall-stones—each of an inch in length and about three-quarters of an inch in breadth, of a pale white colour, and possessing dark greenish-black nuclei of inspissated bile—were enveloped in a compact common capsule of white cholesterin. The extreme measurements of the capsule, judging by my eye, which in such cases is usually to be depended upon, being two inches and a quarter in length, and one inch and an eighth in breadth.

On one occasion a medical gentleman brought to the Physiological Laboratory of University College a gall-bladder containing besides bile a large number

of dark round hard granules. The largest of which was not much bigger than a pin's head, and looked like what sportsmen call sparrow-hail or dust-shot. While the smallest of them were invisible to the naked eye. When a drop of the bile was placed in the field of the microscope, even the smallest of the granules were at once recognised to be perfect little miniature globular gall-stones. Not particles of inspissated bile, but true calculi—I dare say there were many thousands of them in the bladder. I thought at first, as they were hard globular bodies, they were concretions of carbonate of lime, merely stained black by bile-pigment; but as they did not effervesce on the application of strong nitric acid, or leave any residue on being calcined, their organic nature was rendered undeniable. While their fatty constitution was at the same time demonstrated by their flame.

Gall-stones, like mere concretions of inspissated bile, form not only in the gall-bladder, but in the biliary ducts. Even in the small ones in the tissue of the liver itself. As is proved by the fact that small gall-stones have, not only once, but again and again, been accidentally found on *post-mortem* examination, by giving a gritty sensation to the knife when making sections of the hepatic tissue.[1] Small though these intra-parenchymatous gall-stones occasionally

[1] I must here allude to a strange and rare form of calcareous deposit in the liver substance, which might, from the gritty sensation it gives to the knife on a section being made, lead the unwary to imagine that the

are, they sometimes produce many of the symptoms of biliary concretions impacted in the large ducts. Namely, pain, tenderness of the liver on pressure, vomiting, and constipation. Without, however, jaundice. For that only occurs when the hepatic or common duct gets stopped up by them.

Sometimes, though more rarely, one stone alone forms in a radicle of the hepatic duct, and, remaining there until it is of sufficient size to completely block it up, not only causes great dilatation of the distal end of the duct, but, by its irritative effects on the surrounding liver substance, induces suppuration.

A case of this kind is reported by Dr. Thomas Cole in the 'British Medical Journal,' February 28, 1880.

A labourer, aged 24, was admitted into the Royal United Hospital, Bath, on November 18, and died on December 19. He was taken with jaundice a year before, having been suddenly seized with pain and vomiting, lasting for three weeks. In August vomiting and pain came on again, and the jaundice returned. The pain left him in about five weeks, but the skin remained jaundiced. His legs were œdematous,

case was one of intra-hepatic biliary concretions, and thereby lead to erroneous pathological deductions. The case I shall relate is one the specimens from which Dr. Bristowe exhibited at the Pathological Society in 1856. It was the liver of a boy, æt. 16, who died from scarlatinal dropsy, of the natural size, but studded throughout its substance with hard gritty deposits of a beef-coloured earthy material. The earthy formation, which was chiefly found in small irregular-shaped groups in the left lobe of the organ, seemed to be seated in the secreting cells.

bowels loose, motions clay-coloured, and the hepatic dulness extended to the umbilicus. The urine was high-coloured and of specific gravity 1019. December 16, severe pain came in the abdomen, and temperature rose to 101·6° F. During the night he had a rigor lasting an hour and a half, and the temperature reached 104° F., and he gradually sank from the combined effects of peritonitis and hæmatemesis.

At the *post-mortem*, the gall-bladder contained two drachms of thick bile. The liver was congested and bile-stained ; in the centre was a cyst, containing half a pint of clear bilious fluid, and a large number of small dark gall-stones. The floor of this cavity was very much thickened ; and there was a layer of tough lymph, like chamois-leather, adherent to it. This was encrusted by a mass of biliary concretions as big as a small Brazil nut. The hepatic duct was normal.

After these remarks it is advisable for me to state that the vast majority of gall-stones are formed in the gall-bladder, their formation being due to the deposition of the less soluble normal or abnormal parts of the bile. Either as a consequence of these ingredients being present in excess, or in consequence of the solvent, whose duty it is to retain them in solution, being in reduced and insufficient quantity. The formation of gall-stones seems to follow exactly the same law as the formation of stone in the bladder. The only difference being, that while in the one case

urinary salts are the ingredients which go to form the vesical calculus (many of which salts, uric acid, cystin, xanthin, and oxalic acid, being actually formed in the liver, while the kidneys only excrete them), cholesterin, and other biliary products, are the ingredients which go to form the hepatic concretions.

As still further bearing upon the etiology of gall-stones, I may mention that, although they are liable to form in almost every constitution, and it is generally considered that they are most frequently met with in gouty persons, they are nevertheless very frequently met with in individuals of the tubercular and cancerous diathesis, either hereditary or acquired.

It may therefore be said that certain individuals are born with a constitutional tendency to form gall-stones, just as others are born with a constitutional tendency to form urinary calculi. And such being the case, the inborn vice in the system must be kept in check or be eradicated before the formation of gall-stones can be effectually prevented.

Before quitting the etiology of gall-stones I would call the special attention of the reader to an important fact which the perusal of the preceding sixteen paragraphs may possibly have suggested to his mind. Namely, that the vice in the system leading to the production of gall-stones might not unphilosophically be said perhaps to lie simply in one of the two proximate elementary factors :—

(*a*) An excessive production of the substances composing them.

(*b*) Their normal biliary solvents—glycocholate and taurocholate of soda habitually present in the bile—being in deficient quantity.

Symptoms and Signs of Gall-stones.

The most characteristic symptom of gall-stones is PAIN, the most visible sign JAUNDICE. Both of these factors are apparently so easily recognisable that it might be thought that in a diagnostic point of view very little would be required to be said about either the one or the other of them. The sequel will, however, abundantly show that such an idea is a gross error. For not only has an immense deal been written about them by a variety of different authors, but it will be found that I shall now add a great deal of important new material to what has already appeared in home and foreign publications.

To begin with, I have to state that the symptoms and signs of gall-stones lodged in the human body are simply *nil* until they create local disturbance, either by impeding the outflow of bile, or by exciting inflammatory action in the tissues with which they are in contact. For example, gall-stones, large or small, located in the gall-bladder, produce, as a rule, no discomfort and no symptoms whatever. While a gall-stone, no matter what its size may be, occluding

the common bile-duct, gives rise to a distinct and well-marked train of constitutional symptoms and local signs.

As soon as a gall-stone becomes impacted in a bile-duct, slight shiverings, occasionally amounting to actual rigors, followed by feverishness and abdominal pain, set in. At the same time there is stomachal and intestinal flatulency, great nervous depression and mental irritability, loss of appetite, and general *malaise*.

Itching of the skin is one of the most intolerable symptoms of impacted gall-stone. The itching is generally worst on the arms and legs. The fingers and toes are not exempt from it. It is occasionally so severe—especially in women—that they scratch themselves until their flesh bleeds. In general the itching only attacks them in bed, but in bad cases it may come on paroxysmally during the day. It is not pathognomonic of impacted gall-stone ; for it equally occurs, though generally in a less degree, in jaundice arising from any other form of obstruction. From which fact it is my belief that the itching is due to the irritative effects upon the periphery of the cutaneous nerves of the bile acids circulating in the blood.

The pain produced by a gall-stone is in general of a spasmodic or paroxysmal character, and is usually situated in the right hypochondrium, about midway

between the xiphoid cartilage and the navel. It is almost invariably aggravated on pressure—even slight pressure—though relieved by gentle rubbing from right to left. It is in general also associated with nausea and retching, with or without vomiting.

The intensity of the pain not only varies greatly in different cases, but at different times in the same case. It may be a mere paroxysmal twinge, or it may be the most excruciating agony, driving the strongest minded man to shed tears like a child, yell like a madman, contort his body like a fool, or lie rolling sprawling and shrieking upon the floor in the throes of despair. This is no imaginary or highly coloured picture. It is exactly what I have myself witnessed, not merely on one but on different occasions. Indeed, to my personal knowledge, so great was the agony that a strong-minded legal gentleman on one occasion experienced, that he was barely prevented from committing suicide by cutting his throat during the intensity of one of the paroxysms.

Little do mothers imagine (or doctors either) that the agonising and apparently unaccountable screams of children in the cradle are occasionally due to the presence of gall-stones. For the sake of those not well versed in their symptomatology, I may mention that in the 'British Medical Journal' of April 22, 1882, Mr. Dunbar Walker relates the case of a healthy-looking child, who on one occasion, when three

months old, cried incessantly for six hours, on whose diaper were afterwards found three small ovoid biliary concretions of a green colour. The largest weighed two grains. Had these stones escaped notice, as they might readily have done, the cause of the child's agony would never have been ascertained.

The pain arising from the passage of a stone through the bile-duct depends much more on the hardness and shape than on the actual bulk of the concretion. A small hard angular stone producing excruciating agony; a large oval soft one only a fractional part of the pain produced by its hard angular brother.

The sudden TOTAL cessation of pain during an attack of hepatic colic usually arises from the gall-stone having abruptly passed from a smaller into a larger duct, or into the intestines. When, for example, a small stone passes suddenly through the orifice of either the hepatic or the cystic duct into the larger common bile-duct (see Plate I., page 113), where there is plenty of room for it to move freely about without pressing on the walls of the duct, the excruciating pain instantly ceases. So also when it passes out of the common duct into the intestines. Prolonged paroxysmal pain may exist without jaundice; for it is only when the gall-stone becomes impacted in the hepatic or in the common bile-duct, that sooner or later there are superadded to the pain

a distinctly yellow or jaundiced tint of the skin and conjunctivæ, high-coloured urine, and pipeclay-coloured stools.

The sudden disappearance of all the signs and symptoms of gall-stones, when none is discovered to have passed in the stools, is in general attributed to the gall-stone, after having occluded the duct, slipping back into the gall-bladder. This of course originates in the erroneous idea that gall-stones can ascend bile-ducts, which is an impossibility from the ducts possessing valves.

Paroxysmal Pain.

As it is quite possible that the question of how the pain of an impacted gall-stone is paroxysmal may suggest itself to the mind of some readers, I may explain it by mentioning that all the gall-ducts, and more especially the large ones, are, like the arteries, supplied, though in a lesser degree, with contractile muscular fibres, no doubt in order that they may assist by rhythmical contractions the flow of the bile; and that they exert this contractile power in a precisely similar way, in order to favour the passage of gall-stones along their canals, seems highly probable.

The bile-ducts, besides being supplied by contractile muscular fibres, are also freely supplied with nerves of both the sensory and motor varieties, and

although the passage of normal fluid bile along the ducts causes no objective sensations, it is a very different thing when either inspissated bile or gall-stones attempt to make their way through the same channels. And still more so when either a mass of inspissated bile or a gall-stone becomes impacted in the canal, and by the abnormal pressure which it there exerts inflames the lining mucous membrane, and thereby renders its nerves acutely sensitive. Exactly in the same way as the nerves in bone become acutely sensitive to all kinds of physical impressions the moment the osseous tissue surrounding them becomes inflamed. In consequence of the nerves of the bile-ducts being thus rendered acutely sensitive to the presence of the abnormal substance, they, by a process of reflex action, stimulate the muscular coat of the ducts to make violent efforts to expel the intruder. And as periods of exhaustion and consequently of compulsory repose always follow periods of super-effort, again in their turn to be superseded by one of renewed activity, the pain induced by gall-stones assumes a distinctly spasmodic or paroxysmal character.

I must not quit the subject of the characteristics of gall-stone pain without calling attention to the fact that it is not always paroxysmal in its character. On the contrary, it may be continuous, and is invariably so when the gall-stones are not trying to escape from the body by forcing their way along the

natural channels, but by making an artificial channel for themselves by directly ulcerating their way into the stomach, intestines, peritoneal cavity; or out of the body even more directly still through an opening in the abdominal parietes.

Amount of Pain no reliable Criterion of either the Size or the Number of Gall-stones.

It is a very prevalent notion that the larger and more numerous the gall-stones are, the greater must necessarily be both the amount of the pain and the intensity of the jaundice. Neither supposition is, however, correct.

As regards the jaundice, it does not depend so much upon the actual size or number as upon the position and shape of the stones. For example, a circular gall-stone, not bigger than a pea, effectively blocking up the common bile-duct, will sometimes produce a fatal jaundice, while a stone as big as a goose's egg, lodged in the gall-bladder, often causes no symptoms whatever. Again, there might be but one single small gall-stone in the whole body, and yet it might cause intense jaundice and speedy death. While 100 or 10,000 much larger ones might be lodged in the body for five or for fifty years, and cause no disturbance whatever. It is upon the position of the stones then, and neither upon their size nor their number, that the amount of pain and jaundice depends.

From the intensity of the pain, taken in conjunction with the depth of the jaundice, it is in general possible to guess the form and size of the stone. Although this is a rule liable, as will be presently shown, to many exceptions. Yet a small stone or a soft steatomatous concretion very seldom gives rise to pain and jaundice anything approaching in severity to that springing from a large stone, or even to that caused by a hard, rugged concretion of inspissated bile. The danger to life arising from gall-stones may in some cases likewise be conjectured from the intensity of the agony, associated with the depth of the jaundice, though even here again there are many exceptions to the rule. For sometimes but little pain is complained of, and there is no jaundice whatever, when a gall-stone is doing deadly mischief by perforating its way out of the biliary appendages.

Pain as a Cause of Death in Gall-stones.

The pain caused by a gall-stone may be so intense as of itself to produce a fatal collapse. The symptoms preceding death being cold sweats, slow feeble pulse, extreme exhaustion, and coma.

Even with but few of these symptoms manifested, sudden death may be caused by the presence of a gall-stone. One of the most telling cases with which I am acquainted is that reported by Mr. Arthur Sargent, of the Bombay Army, in the ' British Medical

Journal' of June 7, 1879, in which a woman suddenly died after being ill for less than half an hour. The body was thin and ill-nourished. Nothing abnormal exhibited itself, but a lump two inches long in the umbilical region, and a stone completely filling up the small intestine, which was tightly stretched over it. The lump was found to be the gall-bladder, pear-shaped, about an inch and three-quarters long, and three-quarters of an inch thick, completely adherent to the duodenum. It showed a longitudinal fissure, through which the gall-stone had ulcerated. The intestine above and below the gall-stone was perfectly natural.

The Dangers of Gall-stones are not always proportionate to the Amount of Pain.

It is a mistaken though very prevalent idea that you may guess the probable amount of danger to life by the severity of the pain produced by a gall-stone. It would indeed be fortunate in a prognostic point of view were this in reality the case. But, alas! I know it is otherwise, and, what is still worse, that the gradual subsidence of acute agony is in some cases but the forerunner of a fatal result. And a knowledge of this fact is oftentimes a most disagreeable one. For what can be more distressing than to have to warn a patient and his friends of approaching danger when the lull in the storm has given them buoyant hopes of a speedy recovery ?

The explanation of this is very easy. And to make it plain I shall suppose an imaginary typical case.

A patient has a gall-stone for several days or weeks, firmly impacted either in the cystic or in the common duct, and while in the former case he has had most excruciating agony, *without jaundice*, and in the latter equally intense pain with jaundice, he all at once expresses himself as feeling comfortable. For after having suffered the torments of the damned, he feels nothing beyond a slight ache in what was formerly the acutely tender spot, and even moderate pressure no longer augments it much. The natural idea of himself and friends is that he is doing well, and will soon be himself again. On the contrary, he is doing very badly, for the stone has only made room for itself by ulcerating a hole in the walls of the duct, and once the ulcerative process has begun no one knows how the case will end. If adhesion takes place between the walls of the duct and intestines, or the stomach, or the abdominal walls, good and well—for there is a chance that the stone will find for itself a safe exit. But still there remain dangers. For in the first place it may, during its course, lay open a blood-vessel and fatal hæmorrhage occur. Or the stone may, after finding its way safely into the intestines, from being too large to pass through them, produce a fatal obstruction. Or the

stone may perforate its way directly into the peritoneum and induce a fatal peritonitis. So that the learned practitioner looks with horror on the *gradual subsidence* of pain in all cases of hitherto manifested acute agony from gall-stones. On the other hand a sudden subsidence of pain he hails with joy ; for that is not in general indicative of ulceration, but of the exit of the stone out of the duct by the natural passage into the intestines.

Diagnostic Value of the Position of the Pain.

As a gall-stone proceeds from the gall-bladder downwards along the duct into the intestine, the seat of greatest pain changes more and more from the neighbourhood of the xiphoid cartilage, first downwards and outwards to three and a half inches to the right of the cartilage, then downwards and inwards towards the neighbourhood of the navel. The angle of union of these two oblique lines indicates the point of union of the cystic and common ducts. The position of the greatest intensity of the pain on pressure indicates the exact position of the impacted calculus. As before said, the often alluded to shoulder pain I have long since ceased to regard as a point of importance in the diagnosis of gall-stones or any other hepatic affection. While the dorsal pain I regard as even less valuable. For dorsal pain is much more common in renal, stomachical, and duodenal,

than in hepatic disease. Although it has occasionally happened that in cases of impacted gall-stone the chief pain complained of has been in the back.

Dangers of Gall-stones not necessarily in Proportion to their Size.

The dangers arising from gall-stones are not, as is generally supposed, in direct proportion to the size of the calculus. A small stone may find its way into the peritoneal cavity, and induce fatal collapse or peritonitis, while a very large one may ulcerate its way into the intestines, there become sacculated, and give rise to no further symptoms, not even so much as discomfort during a long period of life, and its actual existence be at last only made known by the autopsy after death. I may call attention to a case of fatal obstruction of the common duct by a large calculus, after many weeks of suffering, related by Dr. Thomas Coles in his paper already cited:—'The patient, aged 34 and very delicate, had had a great many attacks, and had passed some very large stones. Every time she became pregnant, calculi were expelled. Her last attack came on when she was five months pregnant. Pain and vomiting were incessant, the jaundice intense, and rigors and sweats most prostrating. Hypodermic injection of morphia, thrice daily, was the only means of relief. At last a stone escaped, another took the place of the one released, and the

poor creature slowly sank, worn out by suffering and exhausted by innutrition, although nourishing enemata had been freely resorted to. At the *post-mortem* examination, a cone-shaped stone was found wedged in the duct, just under its narrowed ending in the duodenum.'

Gall-stones of large size have been found in the stools when not expected. A case presumably of this kind was brought before the Pathological Society on the 6th January, 1880, by Dr. Ord. The stone was sent to him for exhibition by Dr. C. Roberts. It had been passed by a lady shortly after her confinement. There had been only two symptoms associated with its passage: excessive pain in the back, and constant diarrhœa of a pale yellow colour. The stone measured one inch and five-eighths by one inch and one-eighth, and weighed five drachms. The concretion was composed of cholesterin mixed with bile-pigment. Another lady, aged 56, after seven days' sickness, pain, and constipation, voided a pyriform-shaped cholesterin calculus which in a dry state weiged 400 grains and measured two and a quarter inches in length, and one and one-tenth in diameter. In reporting this case (Pathological Society's 'Transactions,' vol. xix. p. 254), Dr. Hilton Fagge mentions that there exists a calculus weighing 462 grains, said to have been passed by a middle-aged lady (along with the fæces), who afterwards enjoyed good health. As there is

no notice of a preceding attack of jaundice having occurred, the stone most probably did not pass through the common duct, but ulcerated its way from the gall-bladder directly into the intestine.

While house physician in the Royal Infirmary, Edinburgh, a rapidly fatal case of jaundice from impacted gall-stone came under my notice. It occurred in a woman, aged 36, who died in Professor Bennett's clinical ward, in the middle of December 1851, and at whose *sectio cadaveris* a pale yellow gall-stone, about the size of a boy's marble, was found firmly impacted close to the duodenal orifice of the bile-duct. The liver was of the normal size, of an intensely green colour, and everywhere throughout its substance the ramifications of the bile-ducts were enormously dilated into elongated cavities big enough to admit the point of the finger, and filled with dark, thick bile. Professor Bennett thought that the patient died from the toxic effects of the absorbed bile, from her symptoms having been vomiting and prostration, feeble and rapid pulse, dry brown tongue, and low muttering delirium; followed by coma and death. There was no remission or intermission of the symptoms in this case. The gall-stone, after having become impacted in the common duct, there steadily remained, producing most intense agony, as well as the above-named symptoms of bile poisoning. From my present knowledge of the effects of reabsorbed

bile, I should say that Dr. Bennett was wrong in attributing the rapidity of this patient's death to bile poisoning ; for when the orifice of the common bile-duct is completely occluded by a cicatrised ulcer, and the bile is absorbed into the blood (*and there is little or no pain*), the patient generally lives for at least eighteen months. So I attribute the death of this patient, which occurred in six weeks after the jaundice had set in, more to the intense pain than to the blood-poisoning.

Danger of Gall-stones not proportionate to the Intensity of the Jaundice they induce.

From the mere intensity or persistence of jaundice *per se*, no trustworthy prognosis can be arrived at. For death may occur in a few hours in a slight case, and recovery actually take place in another after jaundice has existed even for two or three years in an intense form. I shall presently relate a case of gradual recovery from impacted gall-stone, in a lady aged 44, who has been intensely jaundiced for more than six years. It may be said, however, that, as a rule, a case of jaundice from complete obstruction to the flow of bile into the intestines usually ends fatally within three years from its commencement. Although under judicious treatment, in the majority of cases, the life of the patient may easily be prolonged for from four to six years. In those cases where patients

live longer than that, it is generally found that although there has been a permanent obstruction in the duct, there have been slight intermissions of the jaundice, from the stone occasionally changing its position, and allowing bile to find its way past it into the intestines.

Gall-stones even of large size may exist without producing Jaundice.

By far the majority of medical men with whom I come into professional contact possess, I find, the fixed idea that in all cases of firmly impacted gall-stones, jaundice *must* occur. At least in one stage of the disease. Such a belief is, as I shall presently show, founded upon the old crude and imperfect data our predecessors possessed of the clinical history of gall-stones. And as this mistake has on more than one occasion, I have found, given rise not only to an erroneous diagnosis, but to defective treatment, I must call the special attention of my readers, in connection with the general clinical history of gall-stones, to the fact that it is absolutely essential, when attempting to make a diagnosis in any doubtful case, to remember that patients may not alone have gall-stones, but be affected with their severest and most dangerous symptoms, without the skin showing the slightest trace of jaundice.

In some cases the gall-stone, or stones—for there

may be many, even hundreds or thousands—remain in the gall-bladder during the whole life of the individual, without giving rise to any disagreeable results, either as regards pain or jaundice. In other cases the gall-stones—and this usually happens when they are small—get into the cystic duct, becoming lodged there ; and although the patient may suffer intense pain in such a case, there is no jaundice. For it is not until the stones have passed down from the gall-bladder into the common bile-duct that jaundice can be induced by them. An illustrative example of this I shall give presently. Meanwhile I may here only further remark that so long as a stone remains in the cystic duct, although it completely block it up, and effectually prevent the bile either entering into or escaping from the gall-bladder, yet, as in this situation it offers no obstacle to the direct flow of the biliary secretion from the liver into the intestines, there cannot be retention and consequent reabsorption of bile. In fact, the presence of the stone in this position, in as far as the biliary function is concerned, only reduces the patient to the state of a person in whom the gall-bladder is accidentally absent ; or to that of a horse, or other animal, in which the absence of the gall-bladder is a normal condition, and in whom the biliary functions are performed without either hitch or hindrance. This is readily understood when it is remembered that the

gall-bladder is a mere passive receptacle or reservoir for the excess of bile secreted during the intervals of digestion, and is not in the remotest degree, as I previously pointed out, an essential organ in the animal economy. So long, therefore, as a gall-stone, by blocking up the cystic duct, only prevents the bile from getting into or out of the gall-bladder, there is not only no jaundice, but no saffron-coloured urine or pipeclay stools. And it is only the presence of hepatic colic, associated with sickness and the clinical history of the case, which leads to the recognition of the symptoms being due to gall-stone impacted in the cystic duct. I may as well, however, also here call attention to the fact that the cystic duct may, and has in some few cases, become permanently occluded by a deposit of carbonate of lime, and the cause of the occlusion been accidentally mistaken for a gall-stone. A case of this kind where the gall-bladder was also filled by the lime deposit, associated with cirrhosis of the liver and hypertrophied spleen, is recorded in the Pathological Society's 'Transactions' of 1856, p. 238.

Intra-hepatic gall-stones, be their size what it may, are, as a rule, unattended with jaundice, and only with subacute pain. The symptoms they give rise to, when they are either large or numerous, are a feeling of dull weight or discomfort, with sudden and sharp stitches of hepatic colic, accompanied with nausea and retching.

Intense Pain from Gall-stones may occur without Jaundice.

From the fact that jaundice is usually supposed to be an inseparable concomitant of gall-stones, when it is absent the pain arising from them is frequently attributed to a great variety of other causes. To wit, gastralgia, intestinal colic, peritonitis, perforation of the stomach or bowels, renal calculi, &c. &c.

Sometimes the pain produced by a gall-stone, though exceedingly acute, is of very short duration, from the stone quickly getting out of a small into a larger duct, where it not only causes no pain, but gives rise to no obstruction ; and from the fact of its not having remained long enough in the small duct to cause jaundice, and not obstructing the larger duct at all, we have the unusual phenomenon of an acute brief gall-stone colic, without the patient ever showing a trace of jaundice.

This arises from the fact that a jaundiced tint of the skin never appears in less than sixty or seventy hours after complete occlusion of the hepatic or common bile-duct has taken place. Though high-coloured urine and pipeclay-coloured stools may appear several hours earlier.

This piece of information is of great importance in a diagnostic point of view. Again and again have I known the pain produced by gall-stones (in con-

sequence of the absence of jaundice) put down as cramp of the stomach, or, more learnedly, as gastric neuralgia. The sudden advent and occasionally equally sudden cessation of the pain favouring this idea. Ay, what is more, the excruciating agony has been mistaken for perforation of the stomach. A case of this kind I shall now relate. But before doing so, as but little attention has hitherto been paid to the pathology of gall-stone pain, or hepatic colic as it is in general called, without *jaundice*, I shall explain its pathology, which to my mind is very simple. Although I must confess it has not always been so. It is, according to my present ideas, this :—

Gall-stones always produce more or less pain, be their situation what it may, when they press against living tissues, be they the walls of bile-ducts, intestines, or anything else.

Gall-stones *never* produce jaundice except when they impede the flow of bile into the intestines through its natural channels. A glance at Plate I. will make this clear. By showing that, by the arrangement of the ducts, a gall-stone can in two situations only possibly intercept the flow of bile into the intestines. First :—If it blocks up the hepatic duct (*b*), it will not only prevent the secreted bile getting into the intestines, but into the gall-bladder. Second :—If it blocks up the common bile-

duct (*d*), it then not only prevents the secreted bile flowing directly from the liver into the intestines, but it equally prevents the stored-up bile finding its way from the gall-bladder into the intestines.

A stone lodged in the cystic duct (*c*), on the other hand, can by no possibility produce jaundice. This, then, is the true explanation of why gall-stone colic may exist without jaundice.

A most impressive case of this kind fell under my notice while I was acting as house physician in the Royal Infirmary, Edinburgh. It has so many important bearings connected with it, seeing that it was mistaken for a case of perforation of the stomach, that I shall relate it in full. And I do so all the more readily, as I am strongly of opinion that had this very case not fallen under my notice, and made such an impression on my mind as to specially direct my attention to gall-stones in the early part of my professional career, it is highly probable that this book would never have emanated from my pen. Trifles are said to mould the careers of men, and I see no reason to doubt that what I witnessed in the case I am now about to relate is the chief cause of my having made liver diseases a special study.

I will tell the story exactly as it happened, and leave the reader to think what he pleases about it.

I happened after the hour of visit to be passing through one of Dr. ——'s wards on my way to the

fever ward on the same floor, when my attention was drawn to the agonised expression on the face of one of the patients to whose abdomen a nurse was doing something. The man's face was the picture of extreme agony. His eyes were starting from their sockets. Drops of perspiration stood on his forehead. Drops of sweat trickled down his cheeks. His hands clutched the bed-clothes. He literally writhed in torture. A more horrid example of human suffering than what he presented when I approached his bedside it is impossible to imagine. I found the nurse preparing his abdomen in order to apply leeches, and I saw it was not her rough scrubbing that produced his pain. For the pain was apparently increased when she discontinued it. On asking what was the matter, the nurse replied, 'It's a case of perforation, and I am going to put on twenty-four leeches.' 'Who ordered the leeches?' asked I. 'Dr. ——.' 'Did he examine the case?' 'Yes, and he says it's acute peritonitis from perforation of the stomach.' 'I don't believe it,' said I, 'for if it was peritonitis the man could never bear your rough handling. Let me examine him.' I put my hand gently on his abdomen. I rubbed it from side to side. I then pressed it down between navel and stomach, and, instead of increasing the pain, the gentle though firm pressure seemed not only to be borne with impunity, but to give relief. I at once turned to the man, and asked, 'Had you ever an

attack like this before ?' No reply did he, however, vouchsafe. He was in too great pain to pay the slightest attention to me. I repeated the question more emphatically. Still getting no reply, I repeated it loudly. Still not the slightest attention was paid to my question. The man was in fearful agony, I knew; but, being determined to get an answer to my question before I would allow the nurse to apply the leeches, as it appeared to me to be a simple case of intestinal colic, certainly not peritonitis, and having already had nearly two years' hospital resident practice, I thought I knew something about the diagnosis of acute peritonitis, of which I had seen many examples. I gave the man a good shake, and asked him to answer my question with a simple yes or no, as that was all I wanted. He now answered in the affirmative, and, that being enough for my purpose, I ordered the leeches to be put away, a large dose of morphia to be given at once, and the abdomen to be fomented with hot water; then turpentine stupes to be applied. I returned to his bedside in about an hour, and found the treatment had acted like magic. For now the man's expression was no longer one denoting extreme agony. His forehead, though still moist with perspiration, had no sweat drops trickling from it. His teeth no longer bit the bed-clothes. His hands lay in calm repose, crossed upon his breast, and being able to reply freely to my questions he in-

formed me that he had once before had a similar attack. Added to which I gleaned from him the all-important fact that the previous attack had been followed by jaundice. This piece of information at once told me that though my treatment was apparently correct, my diagnosis was probably wrong. For now the case was much more likely to be one of gall-stone than intestinal colic. The only thing that puzzled me was that he had not only no jaundice, but not even so much as a tinging of the skin. I ascertained also that his stools were dark-coloured, and the urine of a perfectly normal hue. If I was puzzled then, I was a thousand times more puzzled a week afterwards by finding him as yellow as a guinea, in spite of his having had no return of the excruciating pain. Pian he certainly had, but nothing to speak of in comparison with what he had when I first saw him. Yet he had jaundice, pipeclay-coloured stools, and bilious urine, all apparently from gall-stones.

The cause of the absence of jaundice in the first instance, in spite of the excruciating pain, coupled with the slightness of the pain and presence of jaundice in the second, was to me a perfect mystery. And for many long years it remained so. For I could find no one who could explain it, and it was not until some years after I had made the pathology of gall-stone affections a special study that I discovered the true mechanism of this apparently anomalous phenomenon.

Which, instead of now appearing to me a pathological puzzle, is easily explicable in the following wise. The man had a stone which suddenly escaped from his gall-bladder into the cystic duct, and, being too large to pass easily through it, caused the agony I described; but, as at the same time the bile secreted by the liver continued to find its way down the hepatic duct (Plate I., *b*), and through the common duct (*d*) into the intestines, there was no jaundice. The morphia I gave him, and the hot fomentations, not only relieved the pain, but helped to dilate the cystic duct and allow the stone to pass into the larger common bile-duct, and from the size of the stone not being great (although it was big enough to block up the duct and induce jaundice by obstruction) it did not again induce excruciating agony like what it did while it was within the much smaller cystic duct.

This case requires no further comments to be made upon it by me. For the reflecting reader must have already taken in all its important differential diagnostic bearings with reference to the question of gall-stone colic in contradistinction to the pain of peritonitis following upon perforation of the stomach, for which it had been mistaken. Chiefly, I presume, from the intensity of the pain, associated with the absence of jaundice. Now, although it is not at all probable that mistakes of this kind can often occur, yet, as there are other forms of colic with which gall-

stone pain is very frequently confounded, so as to lead to grave errors in treatment, I consider the subject of sufficient importance to induce me to devote further on a special chapter to it, under the title of the differential diagnosis of colics.

Gall-stones may be impacted in the Common Bile-duct without Jaundice.

I have next to call attention to what at first sight appears to be a strange fact. Namely, that a gall-stone may be firmly impacted in the common bile-duct, and yet totally fail to induce jaundice, and that, too, not so much on account of the size as on account of the peculiar shape of the stone. As a specimen now lying on the table before me testifies. This stone was passed by a lady (aged 34, the wife of the editor of one of our daily newspapers), after a period of excruciating agony, accompanied, however, with but a very trifling discoloration of the skin. The peculiar form of the concretion, though it is the size of a hazel-nut, amply accounts both for the agony and the absence of jaundice. Paradoxical though this statement at first sight appears to be, it is readily explicable by the fact that the stone is not only triangular, but almost equilaterally triangular. It possesses five sharp projecting points, which accounts for it sticking so fast in the duct, as well as producing so much pain, notwithstanding that it allowed the

bile to ooze past it into the intestines, and thus set all the rules of positional diagnosis at defiance. Here, then, is a case illustrating how a gall-stone may be impacted in the common bile-duct, and yet there may exist neither the jaundiced skin, pipeclay-coloured stools, nor saffron-tinted urine so characteristic of ordinary cases of stone impacted in this duct. Had I been prepared with this knowledge beforehand, the exact position of the stone in the duct (although not preventing the passage of the bile into the intestines) might have been correctly surmised from marking the exact spot of the greatest intensity of pain produced by careful local manual pressure.

Gall-stones as an exciting Cause of Abscess and Cancer of the Liver.

Gall-stones, like inspissated bile, occasionally induce hepatic abscesses as well as even cancer of the liver, but in order to avoid repetition I shall defer the consideration of that branch of their pathology until I come to the special chapters on abscess and cancer of the liver. Meanwhile I shall describe another new phase of the pathological effects produced by gall-stones. Namely, those they induce while attempting to ulcerate their way *directly* from the biliary appendages, as well as those they give rise to in the regions into which they migrate.

Gall-stones passing by Ulceration into the Intestines. Symptoms of Perforating Gall-stones.

When gall-stones ulcerate their way from the gall-bladder into the bowels, there are no well-marked characteristic symptoms. But the practitioner ought to be alive to the fact that when, after hepatic symptoms have existed for a time, there is a considerable increase of the dull aching pain complained of in the region of the gall-bladder upon the application of pressure, and blood appears in the stools, the case is almost for a certainty one of perforating gall-stone. I shall relate a case showing, meagre though these data be, how valuable they are ; for, as will be seen, I was able, by paying careful attention to them (fourteen years before the autopsy revealed the truth of my statement), to correctly diagnose a case of the kind where another consultant, vastly my superior in experience, totally failed. As no doubt the reader will be glad to learn how I came to form a correct diagnosis when another more experienced, placed under precisely similar circumstances, failed, I may as well at once tell him that my talisman is very simple. Namely, that whenever bloody or grumous stools appear in the course of a case with obscure gall-stone symptoms, even without a single gall-stone sign having so much as manifested itself (according to my opinion), the case may be unhesitatingly regarded as one of perforating gall-stone. At

the same time I must remark that the absence of bloody or grumous stools under precisely similar conditions in no wise of itself negatives the idea of intestinal perforation. For perforation sometimes takes place without any signs of hæmorrhage appearing. As the following case related to me by Dr. Leared shows. A woman was suddenly seized with pain in the abdomen and sickness, both of which lasted but a very short time, when thirty hours afterwards, to her surprise, she found a stone as big as a pigeon's egg in her fæces. On examination it was found to consist of almost pure cholesterin.

I shall now adduce two typical cases (one which ended fatally, the other which got well) occurring in women, both of which respectively, though in totally different ways, present all the most salient features usually met with in such cases. The first I shall select from Dr. Peacock's, the second from my own practice.

Dr. Peacock's case was that of a woman aged 27, who was only seriously ill during four days. The abdomen became tense and tympanitic on the third. Collapse supervened on the fourth, and in a few hours afterwards she died. At the *post-mortem* the gall-bladder was found adherent to the curvature of the duodenum, and an aperture big enough to admit the forefinger existed between the fundus of the gall-bladder and the intestine. In which was found a biliary

calculus too large to pass through the duodenum. A somewhat smaller calculus, however, must have done so, as it was found in the ileum. The opening from the gall-bladder into the intestine had been unattended with any great disturbance of health, and it was only when the stone blocked up the intestine, four days before the death of the patient, that really serious symptoms began.

The case I saw possesses several important features, from the fact of its not only affording a typical illustration of the difficulties sometimes encountered in diagnosis, and how the effects of a perforating gall-stone may be mistaken for those of a cancer, but how, in spite of the patient getting well, the diagnosis may be confirmed even to its minutest details by a *post-mortem* made fourteen years afterwards. The history of the case is briefly this. On March 22, 1864, just as I was starting to the country for an Easter week's holiday, I was asked to see a dangerous case along with Mr. Pearse. The patient was the wife of a celebrated ecclesiastic. She had been previously seen by my colleague Professor Walshe, who confirmed the diagnosis of her ordinary medical attendant. Which was that it was a case of malignant disease of the liver. She was so ill that the night before I was summoned to her bedside she had bid adieu to her relatives, and assuredly a less hopeful-looking case than what she appeared to be

when I entered the room it is scarcely possible to imagine. For the poor woman was haggard, bloodless, and worn to skin and bone. Too weak to raise herself in bed. With a voice so feeble as to be scarcely audible. She was jaundiced. The stools were pipeclay-coloured, with the exception of once or twice when they were mixed with blood. Pulse 120, and feeble. The tongue very foul, and breath smelling disagreeably.

I examined, or should rather say that I tried to examine, the liver by percussion, but she was so weak, and it was so painful, that I had to desist after a very imperfect examination of it. So I listened to the history of the case, and examined the urine. Which was literally black, thick, and muddy, containing both albumen and blood-corpuscles. Although it contained a copious deposit of urates, the actual amount of uric acid was supposed to be diminished (I say supposed, because a correct quantitative analysis could not be made from the patient being too weak to admit of its being all collected). The stools contained grumous blood. The liver, as far as could be made out, appeared to be greatly enlarged and tender to pressure. The most tender spot, however, was, I imagined, at the point where the bile-duct would open into the duodenum. When the abdomen was pressed at this particular spot, intense pain was the result. That fact, coupled with the other conditions

above given, led me to diagnose the case as one of gall-stone ulcerating its way into the duodenum. On explaining to Mr. Pearse the reasons which led me to take this view of the case, and particularly when I said I thought the patient would get well, he suggested that I should tell this to the reverend doctor himself, adding, 'You know he is learned in science as well as in theology, so you may unhesitatingly speak to him not only fully, but quite openly.' This I accordingly did, and it was arranged that, if I did not hear to the contrary, I should call that day week, and see the patient on my way home from the railway station. Mr. Pearse accordingly altered the treatment; for, as he justly remarked, as the patient was evidently dying, there could be no possible harm in treating her for gall-stone, and giving her even the faintest chance of recovery. Hot fomentations were consequently had recourse to, an alkaline mineral purgative mixture given, and nourishing stimulating foods prescribed. An immediate change for the better was observable. Rapidly the patient rallied. In a week she was able to sit up in bed. In a month she was on the sofa, and within three months more she was walking about apparently quite well. And for fourteen years afterwards well she may be said to have remained, till at length another gall-stone again ruptured the bile-duct, and in a few brief hours terminated her existence by hæmorrhage and

fatal collapse. The account of her death and *post-mortem* I received from her husband in a letter dated May 9, 1878. Which I shall here give exactly as it stands. With the exception of omitting some family details. For, although written by a clergyman, it is sufficiently pathologically explicit for what it is desired to prove.

'As you took a great interest in the ailment of Mrs.——, I think perhaps you would like to know the revelation of the *post-mortem*. We had three medical men at the examination, and I apprehend their judgment is conclusive. They found three large gall-stones in the bile-duct. The largest was the size and shape of a knuckle-bone, the two above it not so large. They were not of a crystal character, but a hard concreted gum, as hard as rock; they had so distended the walls of the duct that they had burst it, and the gall had made its way out of the chink into the bowels. One other consequence of this enormous distension was the adhesion of part of the bowels to the duct, but the immediate cause of death was the rupture of the duct. Mrs. —— had been ailing since last November, but was considered convalescent. She had been out in the village and garden the day before, and on the very night of her decease we had friends to spend the evening with us. Mrs.—— was with them till about nine o'clock, and then told me she did not feel well, and that she

should go and lie down. About four hours after she died in a tranquil sleep.'

Nothing could be more conclusive than this report. For it proves in every individual particular the exactitude of the diagnosis made nearly fifteen years before. As some of my readers may like to hear what were the chief signs and symptoms in the case which led me to diagnose perforating gall-stone, I may say that what I most relied upon as being indicative of a gall-stone ulcerating its way directly from the common bile-duct into the intestines were (*a*) the indefinable soreness in the right hypochondrium ; (*b*) acute pain on pressure over the tender spot ; (*c*) presence of jaundice ; (*d*) bloody stools ; and (*e*) febrile symptoms, with great prostration of strength, almost amounting to collapse.

Gall-stones that have ulcerated their way into the Intestines may become encysted there.

A case of gall-stone encysted in the intestinal canal and producing no irritation whatever fell under my notice in 1856, and I reported it, under the heading of hepatic intestinal calculus, in the eighth volume of the Pathological Society's 'Transactions,' p. 235. The following is an abstract of its history. The body of a man aged 87, who died in the St. Pancras Workhouse, was being dissected at University College, when Mr. Jakins, one of my then students, found a large

hard oblong mass, measuring three inches in length, and three and three-quarters in circumference, lying in an artificially formed *cul-de-sac* in the right side of the duodenum about equidistant from the pylorus and the entrance of the bile-duct into the intestine. The outer wall of the *cul-de-sac* was firmly adherent to the gall-bladder by old-standing adhesions. The gall-bladder itself was small and contracted, and the common duct abnormally dilated. All these facts when taken together left no doubt that the stone had ulcerated its way from the gall-bladder into the intestine, and thereafter become encysted.

The stone, or it might rather be called stones, weighing (when dried) four hundred and fifty grains, consisted of three distinct portions, articulated and agglutinated together. And as on the middle one were two distinct large lateral facets, it must have been originally composed of five pieces. Judging from the size of the facets, it must have weighed not less than two thirds more than it now did, thereby making the enormous total of seven hundred and fifty grains. On analysis it yielded

Cholesterin . . .	90·346
Mucus	2·218
Pigment and resin . .	4·242
Inorganic salts . . .	0·661

From this analysis it appears that it was a biliary

calculus, although one containing not only less than the average amount of cholesterin, but less than the average amount of inorganic salts. It was no doubt fortunate for the patient that the stone remained lodged in the *cul-de-sac*, for had it attempted to descend through the intestine, from its large size it would undoubtedly have completely blocked up the canal, and produced fatal ileus. The appearance of the exterior of the calculus, as well as those of the old adhesions by which it was surrounded, rendered it probable that it had been lodged where it was found for many years. On making inquiry at the workhouse, nothing could be ascertained of the man's clinical history, except that during the five years he had been an inmate of the institution he had never had jaundice, or been known to complain of any hepatic derangement.

A number of biliary calculi which had escaped from the gall-bladder by ulceration of its coats were found by Dr. Sidney Coupland, at the autopsy of the patient, encysted in peritoneal adhesions close to the pylorus. The case was that of a man aged 40, who died at the Middlesex Hospital under Mr. Hulke's care, from the effects of a tumour growing from the base of the skull. Nothing was known of his liver history, so that there is nothing to add to the fact that gall-stones may become encysted even in the peritoneal cavity itself.

In the Museum of St. Thomas's Hospital there is a preparation with two calculi lodged in an ulcer at the fundus of the gall-bladder, and seventy-five more are said to have been found embedded beneath the abdominal muscles outside of it.

The Danger arising from Gall-stones continues after their Entrance into the Intestines.

That the dangers attendant upon gall-stones do not necessarily cease after they have reached the intestinal canal in perfect safety is a point never to be lost sight of. For not only may they become impacted in some part or other of the intestines, and give rise to distressing symptoms, but their impaction may, and frequently does, lead to fatal results. Many cases of this kind have been reported both in home and foreign journals. Many years ago Dr. Vanderbyl exhibited to the Pathological Society the parts in a case of this kind, where the patient died from ileus caused by a gall-stone.

Mr. Le Gros Clark has put on record, in the 55th volume of the 'Medico-Chirurgical Transactions,' a fatal case of impaction of gall-stones in the ileum in a lady aged 58, who never had jaundice, but eight months previous to her death was seized with pain in the right hypochondriac region (where a hard tumour could be felt), bilious vomiting, and griping pains in the bowels. The fatal attack began with obstinate constipation, and in a few days she was attacked with severe ab-

dominal pain, chiefly in the region of the ileo-cæcal valve, where a hard tumour was felt, and with bilious vomiting which shortly afterwards became stercoraceous. She died in eight weeks from the commencement of this attack without ever having once had a proper motion.

The *post-mortem* examination revealed two large gall-stones blocking up the ileum close to its valve. Each stone measured about four inches in circumference, and one in length. Together they weighed nearly 600 grains. As there was no previous jaundice, these stones must have ulcerated their way from the gall-bladder into the intestines, yet no adhesion or cicatrix was anywhere found.

Another interesting fatal case is recorded by Mr. Lammiman ('British Medical Journal' of May 20, 1876), as follows :—

A woman, aged 54, complained of constipation and some abdominal pain. An ordinary purgative was prescribed ; next morning, there having been no relief, another aperient was given. Early on the third day, abdominal pain increased by pressure, and other signs of inflammation, supervened ; vomiting of a stercoraceous character came on, when she began to sink, and died. The inspection showed the ordinary conditions of diffuse peritonitis, with trifling effusion. The ductus communis choledochus and gall-bladder could not be found, but in their place was a dense

cicatrix connecting their normal site with the duodenum, while in the lower part of the ileum was tightly impacted a biliary calculus, two inches long, one inch and a quarter in diameter, three inches and a half at its greatest girth. It weighed four drachms.

In another fatal case reported by Dr. Baly, the stone, though measuring only one inch in length, had a circumference of $3\frac{3}{8}$ inches.

Gall-stones may induce Enteritis.

It is no uncommon thing for gall-stones, even when small, to become lodged in the ileo-cæcal valve and produce great irritation, but as it is rather an unusual thing for them to induce a fatal result, I shall relate a case of this kind of which the history was given to me by Mr. Ward. The patient (a lady, aged about 40), in whose cæcum after death from an acute attack of enteritis a number of large gall-stones were found, had for some years previously suffered from vomiting, diarrhœa, and great abdominal pain. Previously to this she had been habitually very constipated. Just before her death the stools were like pea soup, both in colour and consistence, with now and again scybalous masses in them, which Mr. Ward thought might in reality have been gall-stones, seeing that a great many were found in the cæcum after the patient's death.

Gall-stones impacted in the Rectum.

Enormously large gall-stones sometimes find their way quite safely through the intestines, and yet become lodged in the rectum. Dr. H. F. Walker (referred to by Dr. Flint at p. 460 of his 'Practice of Medicine') removed from a patient's rectum a gall-stone which measured three and a half inches in its longest and one and a quarter inches in its shortest diameter. In this case the patient had previously suffered from symptoms of peritonitis, which were probably caused by the stone ulcerating its way from the gall-bladder into the intestines. Most likely into the colon. For I hardly think so big a stone could have passed though the small intestine of the patient, seeing it stuck in his rectum, the calibre of which would of course be considerably greater than that of the small intestines.

Gall-stones may be vomited.

Gall-stones are sometimes expelled by vomiting, probably in consequence of their finding their way from the duodenum into the stomach, by the same retrograde peristaltic action of the bowel as leads to bilious and stercoraceous vomiting. Or the gall-stone may ulcerate its way directly from the gall-bladder (by adhesive inflammation and ulceration) into the stomach, and produce so much irritation in its

transit as to lead to the death of the patient. Fortunately, however, this is not always the case, and the stone occasionally passes direct from the gall-bladder into the stomach by ulceration with so little disturbance that its existence is not even so much as suspected until it is expelled by the mouth. A remarkable case of vomited gall-stone I was requested to report upon to the Pathological Society (vol. xii. p. 129). It occurred in the practice of Mr. Jeaffreson. A lady aged 94, after only two days' illness (consisting of pain in the stomach, accompanied with vomiting), ejected a hard dark oval mass, measuring an inch in length and half an inch in diameter, and weighing sixty-five grains. Which on chemical examination I found yielded over ninety per cent. of pure cholesterin. The concretion was therefore a genuine gall-stone. So little disturbance to the patient's health either preceded, accompanied, or followed the passage of the stone from the gall-bladder into the stomach, that beyond the two days' illness she never made any complaint which could be attributed to the presence of the stone. No sooner was it ejected from the stomach than the pain and vomiting ceased; and although six months had elapsed since this occurred, before it was sent to me for examination, she had remained during the whole of that time in the enjoyment of perfect health. So that we are compelled to suppose that if the stone

did not reach the intestines before it arrived in the stomach, the opening through which it passed from the gall-bladder into the stomach must have rapidly healed up. It is of course almost out of the question to imagine that such a large mass could have passed through the pylorus into the stomach from the duodenum by a reversed peristaltic action of the bowel with so little pain, for apparently it was too large to pass easily through the pyloric sphincter. While it would be equally contrary to probability to imagine that it could pass through the narrow orifice of the bile-duct into the duodenum without causing jaundice as well as paroxysmal and excruciating agony. Indeed, the only comparatively painless way it could get from the gall-bladder into the stomach was by direct ulceration. Hence I adopt that theory in preference to any other.

Gall-stones may perforate the Abdominal Parietes.

It is no uncommon thing for gall-stones to ulcerate their way out of the body through the abdominal parietes. Even in very aged people this may happen. For example, a bishop at the age of 90 passed three gall-stones by his umbilicus, the largest of which weighed 180 grains. A woman aged 60, who had twenty-two years before suffered from jaundice, passed, after great pain, by an ulcerated opening close to the navel, a biliary calculus an inch and a half in length

by three-quarters of an inch in diameter. The opening rapidly closed, and the woman got quite well. The case occurred in 1852 at St. Thomas's Hospital, under the care of Mr. Simon.

Dr. J. W. Ogle in 1854 reported the case of a man at the age of 57, who passed about thirty gall-stones of the size of small nuts through an ulcerated opening in the umbilicus, and who, after their discharge, got quite well, and subsequently (ten years later) died of phthisis. At the *post-mortem* one small biliary calculus was found embedded in the diaphragmatic surface of the right lobe of the liver. The omentum was adherent to the umbilicus, and in it could be made out the boundaries of an abscess which had evidently originally implicated the gall-bladder, as it contained a quantity of dark-coloured liquid in which were found a number of agglomerated biliary concretions composed of mucus and inspissated bile, altogether making a mass of about the size of a hen's egg.

Another case of an analogous kind is recorded by Dr. Robertson in the Pathological Society's 'Transactions,' vol. v. page 158, in which a man aged 67 passed about thirty gall-stones the size of nuts. During the time the fistula at the umbilicus remained open, the discharge of yellow matter from it was so excessive that it brought him to the very point of death. It ultimately healed up, and the man

lived for three years afterwards. At the *post-mortem* one small irregular biliary calculus was found embedded in the diaphragmatic surface of the right lobe of the liver, but none elsewhere. The gall-bladder was shrunken, but contained a small quantity of bile.

That even attacks of gall-stones or colic without jaundice may be followed by the establishment of a biliary fistula is well shown by the case of a lady, aged 40, who was under the care of Mr. Curling. For many years she had been subject to paroxysmal pain in the hepatic region accompanied with vomiting, but never any jaundice, when suddenly a swelling was noticed immediately below the margin and about the middle of the right ribs. It gradually increased in size and diminished in hardness. At length fluctuation made its appearance, and it was lanced, when several ounces of viscid yellow but not bilious-looking fluid came away. Within a month of the date of the evacuation a biliary calculus not bigger than a hempseed came through the fistulous opening, and three months later four slightly larger ones were also discharged, which was immediately followed by a diminution of the pain. Two days afterwards, however, the pain returned in an agonising form, especially in the back, with violent sickness, and all at once an unexpected discharge of pure dark-green bile took place, and continued at the rate of from one to two ounces in the hour—being most after meals—for nearly two

months, when, after the expulsion of another equally small biliary concretion, the wound closed. Dr. Murchison (Pathological Society's 'Transactions,' vol. xxii, page 153) gives the *rationale* of the case as follows:—

(*a*) A concretion which had been formed in the gall-bladder entered the cystic duct, causing paroxysms of pain and vomiting, but no jaundice, as the common duct remained free.

(*b*) The cystic duct being blocked up by the concretion, no more bile could obtain access to the gall-bladder. The bile already retained there became absorbed and replaced by a viscid opaque fluid. The gall-bladder became distended and formed the tumour midway between the margin of the ribs and umbilicus in the perpendicular right nipple line, which was opened, and its contents evacuated along with five biliary concretions.

(*c*) A fresh attack of biliary colic followed. The concretion already spoken of as blocking up the cystic duct became dislodged, passed down into and obstructed the common bile-duct. The bile could then no longer pass along it into the intestine, therefore passed by the now free cystic duct into the gall-bladder, and from thence discharged itself through the fistulous opening in the abdominal parietes.

(*d*) With another attack of biliary colic the stone was again dislodged, and passed through the common bile-duct into the intestines. The duct thus being

once more free, the secreted bile passed along it into the duodenum and not into the gall-bladder, and, there being no further necessity for the fistulous opening between the gall-bladder and abdominal parietes, it healed up and the patient got well.

Gall-stones may ulcerate their way into the Urinary Organs.

Biliary concretions have been known to ulcerate their way into the pelvis of the kidney and even into the urinary bladder.

Güterbock describes in 'Virchow's Archiv,' vol. lxvi., a biliary concretion which had ulcerated its way into the urinary bladder, and was removed by lithotrity from a woman aged 50, who had observed no other symptoms than those produced by the presence of the calculus. An examination showed the concretion to consist of cholesterin, with small quantities of urea, phosphate of lime, and bile-pigment. The urine had contained bile-pigment. The concretion removed weighed in all 200 grains. The author refers to two recorded cases of gall-stone in the urinary bladder. In one, there was found an obliterated communication between the gall-bladder and urinary bladder through the urachus, in which there had been a temporary discharge of biliary colouring matter with the urine.

Biliary calculi, after finding their way by ulceration into the pelvis of the kidney, have passed down

along the ureter with all the symptoms of ordinary renal colic, become for a time lodged in the urinary bladder, and been ultimately voided along with the urine, and their true nature and clinical history only been revealed by their chemical analysis.

A still more remarkable case is related in the 'Gazette des Hôpitaux,' Paris, October 8, 1846, where two gall-stones were removed from the pubes of a female, which had passed down along the recti muscles and become encysted in the subcutaneous tissue a little above the clitoris.

Gall-stones may cause Death by Hæmorrhage.

Gall-stones sometimes cause death by ulcerating their way into a blood-vessel. This is well shown in a case (reported by Dr. Bristowe at page 285 of the ninth volume of the Pathological Society's 'Transactions') of a woman aged 32, in whom after death it was found that the common duct was not only obstructed and dilated by a gall-stone, but suppuration had occurred, and a communication been established between the bile-duct and the portal vein, through which bile had flowed and mingled with the blood. She had suffered from intense jaundice, with all the symptoms of obstruction to the flow of bile into the intestines, and in addition presented just before her death the unusual spectacle of spitting up pus stained with bile. This was partially accounted for by the

discovery in the liver of cavities filled with bile-stained purulent fluid, which communicated with the lung through the medium of the vena cava.

Fatal hæmorrhage may even arise from a rupture of the gall-bladder taking place on account of occlusion of the common bile-duct by an impacted gall-stone. Dr. Leared recorded a case of this kind which occurred in an otherwise healthy young man aged 22 (*vide* Pathological Society's 'Transactions,' 1859, p. 177).

Decided Symptoms of Gall-stones, yet not arising from Gall-stones.

All the symptoms of a gall-stone may have existed in a marked degree, and then disappear—the skin get pale, the stools of a natural colour, and the urine cease to be bilious-looking, as in a case of gall-stone—and yet no stone, or even any form of a biliary concretion whatever, have been present. And why? Simply because the common bile-duct may become plugged up by other things besides gall-stones or inspissated bile.

For example, hydatid cysts, as well as intestinal worms, have been observed to block up the bile-ducts. An interesting case of jaundice occurring in this way in a girl aged 16, who died after a few weeks' illness, where death resulted from the presence of hydatids in the ductus hepaticus and

ductus communis choledochus, is reported by Dr. Dickinson in the Pathological Society's 'Transactions' (vol. xiii. p. 104).

A similar case fell under my own notice; but, in order to avoid repetition, I shall delay giving the history of it until I come to the special chapter on hydatids. Meanwhile I shall cite one that was published by Dr. Becker in the Berlin 'Klinische Wochenschrift,' July 14, 1879, in which there existed not only jaundice with an enlarged and tender liver, but both pain and vomiting, and consequently it resembled in every individual respect an ordinary case of obstructive jaundice from gall-stone. It was all the more easily mistaken for gall-stone, as the jaundice came and went, and the stools were only pipeclay-coloured and the urine of a dark tint during the attacks. On one occasion the liver was so greatly enlarged as to extend nearly into the pelvis. All at once the lad, who was 17 years of age, began passing portions of echinococci in his stools, which, when placed under the microscope, revealed numerous hooklets. Dr. Becker calculated that he passed about 500 hydatid vesicles in the course of ten days! After which all the jaundice signs and symptoms left him, and he improved rapidly, but only for a time; for soon afterwards he began to have a suffocating kind of cough, and to vomit tough stringy mucus and about a teacupful of bile, in which were five hy-

datids as large as walnuts. The vomiting continued for several days, during which time it was estimated that he brought up about fifty more. On one occasion the vomit was of a greenish purulent colour and contained small clots of blood, and at the same time the characters of the expectoration were such as to lead to the idea that the hydatids had burst into the bronchi. The patient died of exhaustion, but unfortunately no necropsy could be obtained.

Cases have been reported where cherrystones are said to have got from the intestines into the common bile-duct and produced jaundice by blocking it up. Such cases, however, are, in my opinion, misstatements arising from small gall-stones having been mistaken for cherrystones. A not improbable supposition, seeing how very closely some gall-stones resemble cherrystones both in size and external appearance. I doubt if cherrystones ever caused jaundice in the manner attributed to them, from the fact that the intestinal valvular orifice of the bile-duct presents a great obstacle to the entrance into it even of fluids from the intestines. And even supposing that one cherrystone by some accidental means or another found admittance into the bile-duct, it is not, I think, at all reasonable to suppose, or even possible to imagine, that a series of them would one after another find their way through the narrow orifice of the duct. The

diameter of whose interior being so small that they are supposed to have afterwards so effectually blocked it up as not to allow any liquid bile to pass between them and its walls, and thereby produce the jaundiced state attributed to them.

Treatment of Gall-stones.

The treatment of gall-stones was, until recent years, regarded by every one as a thankless task; I think my readers will have, however, already learned from the results recorded in some of the cited cases, that it need be considered so no longer. The difficulties in the path of their treatment vanished one by one with the advent of improved methods of diagnosis. Once the presence of a gall-stone has been satisfactorily made out, and its situation as well as its mode of attempted exit clearly ascertained, its treatment is, comparatively speaking, an easy matter. It may be a bold, but nevertheless it may not be an untrue, thing to say that the dangers arising from gall-stones are in direct proportion to our want of an exact knowledge of their whereabouts. For once their position is correctly known, a pretty fair guess can be made not alone at what they are doing, but also as to how best we can assist them in making a safe exit from the body.

It is with gall-stones as with urinary calculi: the earlier they are diagnosed, the easier is their treat-

ment. In all cases prevention is better than cure; and as I have clearly pointed out the constitutions and habits of the persons in whom they are most likely to occur, I shall begin by saying a few words on

The Prevention of Gall-stones.

As every enlightened practitioner now knows that there is nothing easier in preventive medicine than to intercept the formation of urinary calculi, it will not surprise any of my readers to hear that it is almost equally easy to prevent the formation of gall-stones. For the very simple reason that, in the vast majority of instances, cholesterin is their chief ingredient, and the abnormal formation of cholesterin can readily enough be prevented by foods and by medicines.

The whole rationale of treatment, as regards foods, may be embodied in a single sentence: 'Let the patient avoid an excess of fatty and fat-forming foods, either liquid or solid.' When I have said this, I have said almost all that is necessary on the subject of diet, for here, as everywhere else, the major includes the minor. Two forms of food I must, however, make special allusion to among the fat-forming. Namely, sugar and starch. These are especially to be avoided, or at least only indulged in with moderation. I am thus particular in making allusion to starch and sugar, from the simple fact that I find that at least one half of the patients who come to me

labouring under gall-stones are not only great butter or bacon eaters, but much given to indulging in saccharine and farinaceous substances.

In the human body not only is starch rapidly transformed into sugar, but sugar into non-crystallisable fat, and that again in its turn into crystalline cholesterin.

If this theory be correct, which I have good scientific as well as clinical data for believing that it is, the line of preventive treatment to be adopted is self-evident. Having struck out from the patient's diet roll the excess of cholesterin-forming materials, the next and only remaining point is to ensure a sufficient supply of glycocholate and taurocholate of soda in the bile to retain all the cholesterin contained in it in a state of fluidity. After what I said in the physiological chapter regarding the chemical constitution and properties of bile, all I need now add upon this point is, that the administration of an excess of alkali in some form or another has the desired result. For the alkali is absorbed and carried to the liver along with the portal blood, there enters into combination with the bile acids, and forms glycocholates and taurocholates, which possess the special and important property of retaining biliary solids in a fluid state ; and although I am not sufficiently sanguine to believe that an alkali can dissolve a gall-stone of any considerable size, by inducing the forma-

tion of an excess of glycocholate and taurocholate, cholesterin solvents, in the biliary secretion, I am nevertheless of opinion that alkalies are of the utmost advantage not only in preventing but in arresting the deposition of gall-stones. The alkali to which I give preference is the carbonate of soda, and the reason why I prefer it to the carbonate of potash is in consequence of my believing that the advantages derived from administering alkalies in cases of gall-stones are, as already said, entirely due to our being able thereby to increase the amount of glycocholate and taurocholate of soda present in the bile. Both of which substances, separately or combined, retain cholesterin in a soluble form; and, as I pointed out, by far the greater number of biliary calculi are composed almost entirely of pure cholesterin. Therefore it is that I believe that we have it within our power, by the judicious administration of soda, not only to prevent the formation, but even to reduce the size of already formed gall-stones. I say this unhesitatingly, as I have on more than one occasion had ocular demonstration of the fact, and have at this moment in my possession gall-stones which incontestably prove it, inasmuch as they have apparently a distinctly eroded external surface from the solvent action of something, and that something must be presumed to have been the glycocholate and taurocholate of soda; as they were passed after the con-

tinued and free administration of alkalies. This is rendered all the more probable, if not even undeniable, by the fact that in one of the cases a gall-stone which had been passed not only before the alkaline treatment was begun, but before I saw the patient, has quite a rough exterior, while those passed after the prolonged administration of the alkalies are, as just said, all smoothly eroded on the surface. Thereby, to my way of thinking, indisputably proving that they had been chemically acted upon in the patient's body by a solvent of some kind or another.

The administration of carbonate of soda has yet another advantage. For, as was long ago observed by Dr. Prout, gall-stones are very common not only in persons of a gouty, but also in those of a rheumatic tendency of body. A fact which I have myself been able in another way to confirm. For on several occasions, on making a quantitative analysis of the uric acid in the twenty-four hours' urine, I have found that the carbonated alkali has been of double service. In a case of gall-stones, in a woman aged 36, where there was an almost daily deposit of fine crystalline uric acid in the urine, it was found that after the administration of ten grains of soda, three times a day during two months, the tendency to lithic acid deposit was entirely overcome.

I frequently prescribe lithia to persons of the

uric acid diathesis in whom I have reason to suspect the existence of a predisposition to gall-stones. Generally giving it in the form of citrate, in from three to six grain doses three times a day.

The Dissolution of Gall-stones.

Dr. M. Schiff, acting upon the theory that I have so long taught—namely, that cholesterin as well as inspissated bile concretions do not entirely arise from an excessive secretion of the materials forming the stone, but from a deficiency of their natural solvents, water and the glycocholate and taurocholate of soda—advises the administration of cholate of soda in cases of biliary concretions. This is certainly a step in advance of my plan, for I only give the soda in the belief and in the hope that it will form the cholate of soda in the system; while he gives the cholate of soda already made. The dose he recommends is eight grains thrice a day. When the system becomes saturated, as it were, with the remedy, the pulse, he says, becomes slow and irregular, and when that occurs the medicine is either to be entirely discontinued or the dose diminished. Of course, in order to produce a solvent action upon a concretion, it is essential that the remedy should be administered continuously for some time.

As is well known to everyone, the more difficult a disease is to cure the greater is the number of 'speci-

fics' suggested for its treatment, so it cannot surprise anyone to learn that iridin is said to be an almost infallible remedy for gall-stones. How it is supposed to act on a solid lump of cholesterin I know not; but in case any of my readers may feel inclined to try it, I may, by way of encouragement, tell them that Dr. P. A. Young, in a letter to one of the weekly medical periodicals in September, 1881, says that he tried it with the view of preventing the formation of gall-stones, and that he found the result most encouraging. He says that he gives pills consisting of iridin gr. j.; pil. rhei co. gr. iv.; every night at bedtime till twelve are taken. If necessary, some saline mineral water in the morning. A course of the pills he gives once in two months.

A few years ago a mixture of sulphuric ether and turpentine was very extensively used, especially in France, as a solvent for gall-stones. This line of treatment was adopted on account of the well-known solubility of cholesterin in sulphuric ether, and it was thought that the remedy would act upon cholesterin concretions in the gall-bladder in the same manner as it did out of the body. After a time, faith in the powers of the mixture became shaken, and it at length gradually ceased to be employed.

Within the last twenty years, Dr. Bouchut[1] revived the same theory with another form of remedy,

[1] *Edinburgh Medical Journal*, 1861, p. 398.

namely, chloroform, which he administered internally, with the view of dissolving any inspissated bile or biliary calculi lodging in the gall-bladder. Dr. Bouchut stated that he had treated one case of gall-stones in this manner with success. Now, although I have not the slightest desire to throw discredit on the statement of Dr. Bouchut, I must candidly admit that I am very much inclined to doubt the accuracy of his observations. For, in the first place, it is not always easy to ascertain with perfect certainty the existence of one or more biliary concretions so long as they remain in the gall-bladder, and it is equally difficult to know, after one or more gall-stones have been passed by a patient, whether or not all have come away. If, then, we administer chloroform to a patient, either before or after a gall-stone has been found in the stools, we cannot, with anything approaching to certainty, attribute the cessation of his symptoms to the circumstance of the chloroform having dissolved the supposed remaining stones. In fact, on physiological grounds, I very much doubt the efficacy of either sulphuric ether or chloroform as solvents of gall-stones in the living body. Sulphuric ether and chloroform would no doubt dissolve a concretion of cholesterin in the gall-bladder, were they admitted into that viscus in a pure state and in sufficient quantity. But we have no proof that such is the case. On the contrary, we know, at least in as far

as chloroform is concerned, that no sooner does chloroform become absorbed, and mingled with the constituents of the blood, than it becomes decomposed, the chlorine combining with the blood, and formic acid being set free.[1] But even supposing that sulphuric ether and chloroform existed in the blood in a free state, they could not possibly do so in a sufficiently concentrated form to be able to act as solvents of biliary calculi.

My own experiments on animals have shown me how rapidly fatal even small quantities of chloroform are when injected into the circulation, and a similar remark is applicable to sulphuric ether. A few drops of these substances can very readily be injected into the circulation with impunity;[2] but the quantity cannot be increased beyond a certain amount—to far less a quantity than could possibly dissolve a single grain of cholesterin; otherwise immediate death would follow the operation, by inducing a state of chloroform or ether muscular rigor of the body, closely resembling what is called rigor mortis, from which the animals never recover. I am, therefore, completely at a loss to understand how these remedies can be of service in dissolving gall-stones in the

[1] Jackson, *Comptes Rendus*, February 25, 1856.

[2] Vide the author's paper on a method of producing diabetes artificially in animals, by the injection of stimulants—alcohol, ether, chloroform, ammonia, &c.—into the portal circulation. *Comptes Rendus de la Société de Biologie de Paris.* 1853.

living body ; and as I make it a rule as seldom as possible to prescribe a remedy without a clear theoretical knowledge of its physiological action, I have not ventured on an empirical trial of the effects of sulphuric ether or chloroform administered internally in cases of gall-stones.

Ether, chloroform, and turpentine have all been put up into beautiful pearl-like capsules by ingenious Frenchmen in order to facilitate their administration, and these are sold in Paris and elsewhere, under the titles of ether pearls, chloroform pearls, and turpentine pearls. I cannot venture, however, to recommend them. Indeed it is my belief that the beneficial effects both of sulphuric ether and chloroform, when administered in cases of gall-stones, do not in the slightest degree consist in their solvent action upon the calculi, but in their anæsthetic influence on the nervous system of the patient, and through it on the paralysing of the bile-ducts. The anæsthetic effects tending not only to lessen the pain produced by the gall-stones, but also to dilate the ducts, and thereby facilitate the passage of the stones. Sulphuric ether, chloroform, chloral, belladonna, and opium, all act, I believe, in a precisely similar manner.

Although I said a good deal about podophyllin, when on the subject of the general treatment of liver diseases, I must return to that drug, and call particular attention to what I consider to be its most in-

judicious employment in cases of gall-stones. For, as I said before, like every other new remedy it is at this very moment running considerable risk of falling into universal disfavour, in consequence of its too sanguine admirers blindly prescribing it in all imaginable cases of hepatic disease, in many of which it is quite unsuitable, if not actually detrimental. For example, as before said, in cases of jaundice, podophyllin is, at one and the same time, the bane and the antidote. The bane in *all* cases of jaundice from obstruction, the antidote in a few cases of jaundice from suppression. Having already indicated (page 178) the cases in which it may be administered with advantage, I shall now proceed to call attention to one of those where it cannot be employed without injury, and one in which it is, nevertheless, frequently given. The case I allude to is that of gall-stones. When once a gall-stone has formed, and is blocking up the common bile-duct, thereby causing jaundice from obstruction, it is easy enough to understand why a substance like podophyllin, which increases the biliary secretion, is to be avoided. It is not, however, so easy to understand why the remedy is equally counter-indicated, either during the formation or sojourn of a gall-stone in the gall-bladder. This point, therefore, I must try to explain.

In speaking of the mode of formation of gall-stones in the gall-bladder (page 555), I stated that

their formation is due to the deposition of the less soluble parts of the bile, either as a consequence of these ingredients being in excess, or in consequence of the solvent, whose duty it is to retain them in solution, being in reduced quantity. It follows, then, as a natural result, that the longer bile sojourns in the gall-bladder, and the thicker it becomes, the more likely are its constituents to be deposited, and increase the size of the already existing concretion, or give origin to a new formation. It may be further added, that the greater the amount of bile secreted, the longer is it likely to remain in the gall-bladder, and, from osmosis of its aqueous parts taking place, the more concentrated will it of course become. For, as is well known, there is a constant absorption of the aqueous particles of the bile going on during the whole time it is stored up in its reservoir. If, then, during the intervals of digestion, the liver secretes merely sufficient bile to meet the requirements of the succeeding meal, by the end of the digestive process the gall-bladder will be entirely emptied of its contents, and ready to receive a fresh supply. Whereas, if the liver secretes more bile during the intervals of digestion than the wants of the system require, after the completion of each succeeding meal the excess of bile will remain behind in the gall-bladder, and, from becoming stored up with that subsequently secreted, will of necessity favour the increase and excite the for-

mation of gall-stones in persons predisposed to them. There being nothing more conducive to the deposition of biliary calculi than a well-filled gall-bladder.

As a warning against the indiscriminate use of podophyllin, I may cite the following case, which came under my notice as the sheets of my first book on jaundice were passing through the press. In it I remarked that I had received a telegram requesting me to visit, as early as possible, a lady dwelling in the neighbourhood of St. John's Wood. On my arrival I found the lady suffering from a well-marked jaundice, and considerably prostrated in consequence of her having just arrived from Brighton, where she had gone for the benefit of her health, but where, instead of getting better, she got considerably worse. The history of the case was, that the lady had been seized with pain in the back (middle of dorsal region) about three weeks before I saw her. That there had been great tenderness in the region of the gall-bladder —so much so, that she could scarcely tolerate the pressure of her stays ; and that she had suffered from occasional attacks of sickness after eating.

On examining the patient I found the liver enlarged, and tender on pressure. The gall-bladder much distended, and easily felt. The skin of a yellow hue. The stools of a pale tint. The urine very dark-coloured, and loaded with lithates. I had, consequently, no difficulty in diagnosing the case as one

of gall-stone impacted in the common bile-duct; but on communicating my suspicions to the patient, I was informed that such could not possibly be the case, for during a considerable time past she had been carefully treated with podophyllin. Indeed, I learned to my surprise that she had taken from a quarter to half a grain of that substance nearly every day during the previous six months.

This incidental piece of information, instead of shaking my opinion, as the patient had apparently expected, only tended to strengthen my suspicions, for the reasons previously given, namely, that the podophyllin must have tended to keep the gall-bladder constantly full of bile. I accordingly prescribed for the case as one of impacted gall-stone, and left instructions that the stools should be carefully examined for its appearance.

On the following day the patient felt better; although the jaundiced tint was deeper, the stools paler, and the urine still very high-coloured. The deposit of lithates had, however, slightly diminished. Still, feeling certain that the case was one of impacted gall-stone, I ordered the medicine to be repeated, and the stools to be again carefully examined. The plan I recommended was to mix the stool freely with water, and either decant the supernatant fluid, and then add fresh portions of water till the whole of the soluble matter was removed, or to strain the mixture through

a hair-sieve. For in either case the gall-stone would remain behind, and be readily detected. On my arrival at the patient's house the next day, the maid met me with an expression of satisfaction which could not be misinterpreted, and I had scarcely entered the sick-chamber when, with an air of triumph, she handed to me a gall-stone about the size of a large garden-pea or small field-bean. It had been passed that morning about eleven o'clock. That is to say, about fifteen hours after the second dose of medicine. On analysis the stone was found to consist almost entirely of cholesterin, and I have not the smallest doubt in my own mind that to the constant use of the podophyllin may, in a great measure, be attributed its formation. Unfortunately the stone had been accidentally broken into fragments before I saw it, and I was consequently unable to ascertain decidedly whether it was a solitary calculus, or one of many. Had it been one of several, it would of course have possessed facets. One facet would have indicated that the stone was one of two; two facets, that three stones existed; three facets, that the gall-bladder had contained at least four calculi; while four or more facets would denote that the stone was one of many; whereas, if it was a solitary calculus, no such markings would be present.

I may merely add, in conclusion, that from the time the stone passed, the stools resumed their

normal colour—the first two or three were much darker than natural, in consequence of the sudden escape of the pent-up-bile—the urine gradually became pale and clear, and the skin regained its wonted hue. The latter change was expedited by the administration of benzoic acid, and in a week from my first visit a stranger would have been quite unable to detect that the patient had laboured under a recent attack of jaundice.

To this report I have now to add that this patient was for two years more or less under my care, during which time she passed other twelve gall-stones, all a little larger than the first one that passed. One even as big as a small hazel-nut. Thus making thirteen stones in all, and since the thirteenth was passed, now nearly twenty years ago, she has never had another attack of jaundice nor any other signs or symptoms of gall-stones whatever.

Mineral waters are often advantageously employed in the treatment of gall-stones. The natural waters of Carlsbad, Vichy, Vals, Harrogate, Friedrichshall, Leamington, and Püllna being most generally used. But as I have already fully explained, at page 215, the therapeutical effects of mineral waters, as well as pointed out their different modes of action in cases of hepatic disease, I need at present say nothing further regarding them.

The Expulsion of Gall-stones.

When the diagnosis has been correctly made, and the symptoms of the presence of a stone in a duct are severe, all our efforts must be directed towards one goal—namely, the safe exit of the gall-stone from the body, by facilitating its passage along the common bile-duct into the intestines, and favouring its expulsion thence along with the fæces, which may be regarded as the natural course. When a gall-stone has already become impacted in a bile-duct, it may be said, as a rule, to have reached a stage of its career when it would be futile for the medical practitioner to merely attempt, by reducing its size by a process of dissolution, to favour its passage along the duct. That may be attempted, but now at least only as an adjunct to the accelerating its passage onwards by obtaining a dilatation of the duct. If the stone be not large and the symptoms are not severe, we can in general successfully aid its passage through the duct by administering an anodyne containing a full dose of the tincture of belladonna (which apparently assists in dilating the duct) and placing the patient in a warm bath ; while during the paroxysms of pain, the occasional inhalation of the vapour from a mixture consisting of one drachm of alcohol, two of chloroform, and three of sulphuric ether, poured on a handkerchief made into the form of a cup, is generally

attended with great relief. Each or all of these modes of treatment may be followed either by a brisk emetic or purgative, in the hope that the efforts of vomiting or purging may hasten the expulsion of the stone. Although opiates, as said at page 237, are not as a rule admissible in the majority of hepatic cases, that of impacted gall-stone is an exception; for when the pain in consequence of its excruciating character is exhausting, the hypodermic injection of morphia over the painful spot ought to be had recourse to. But in no case is the administration of belladonna to be omitted, and when given along with an opiate the administration should be repeated every two hours or so until the physiological effects of both remedies are produced. When giving opium along with belladonna, I usually select either the tinct. camph. co. or the tinct. opii ammon., on account of both of these preparations containing benzoic acid; for, as shown in the chapter on general treatment, benzoates are beneficial, and it is always wise policy to try and kill two birds with one stone.

I need only further add, as regards belladonna, that it can be equally usefully employed in the form of liniment, plaster, and suppository. Gentle friction with the warm hand from right to left, but not from left to right, over the seat of acute pain, together with kneading pressure downwards from gall-bladder towards navel, may also be had recourse to; and

during the whole time this treatment is being followed out, let the sufferer drink freely of alkaline warm water. A teaspoonful of bicarbonate of soda to the tumbler of warm water, or milk and water, is what I usually recommend. It is impossible, I think, to give too much of this alkaline drink, for it soothes in more ways than one. First by relieving the irritability of the stomach, secondly by facilitating the vomiting, thirdly by favouring the secretion of liquid bile, a copious flow of which is favourable to the floating out of the stone. The administration of a brisk alkaline mercurial purgative ought under no circumstances to be omitted. The form I give (of course in varying proportions to the circumstances of the case) is—

Pulv. Hyd. c. Creta . . .	gr. viij.
Pulv. Rhei	gr. iv.
Magnesiæ	℈ j.
	M.

Olive oil is frequently prescribed in order to facilitate the passage of gall-stones through the intestines, and with good effect. Dr. Austin Flint relates the case of one of his patients ('Practice of Medicine,' p. 460) who, after taking a pint or more for several days, got 'rid of a large number of stones without pain;' and he refers to a case quoted by Dunglison, in which olive oil not only relieved the pain, but was followed by the discharge of a number of

globular fatty-looking masses varying in size from a pea to a grape, which 'cut with a knife like soft wax.' No doubt steatomatous concretions.

Treatment of Head Symptoms.

When head symptoms supervene in the course of an attack of gall-stone colic, I have found decided benefit accrue from the administration of repeated doses of

℞ Ammonii Chloridi . . gr. xx.
Pulv. Antimonialis . . gr. iij.
Aq. Sambuci . . . ʒ ss. M.

Large doses of liquor ammoniæ acetatis are likewise exceedingly useful in cases of delirium from biliousness. The stage in a gall-stone's career which, on account of the signs and symptoms, is not alone the most alarming to the patient and his friends, but also the one which usually causes the greatest amount of anxiety to the medical attendant, is when it is impacted in the common bile-duct. For one can never feel quite certain what ultimate course the stone will take, and consequently whether or not the case may come unexpectedly to a fatal termination.

Treatment of Firmly Impacted Gall-stones.

When a gall-stone has become firmly impacted in the common bile-duct, the majority of practitioners

regard the case as a hopeless one, and usually reckon the patient's life by months. Fortunately I believe that I am in a position to state positively that almost no case of impacted gall-stone is hopeless, for my own experience tells me quite the reverse. I have known, and at this very moment know, patients who have had gall-stones firmly impacted in their ducts for years, not only without dying, but ultimately recovering. And their treatment has not been difficult, seeing that it essentially consisted in only two particulars—dilating the duct by belladonna, and reducing the size of the stone by appropriate solvents.

As an encouragement to my readers I shall relate three cases which have more than usual interest attached to them. The first is one I saw along with Surgeon-General Dr. George Smith, I.M.S., and which I will give in his own words.

'A. B., born in India in 1855. In June 1877, had an attack of hepatitis. Health was quite restored after the attack. Took much exercise, and seemed to be in perfect health. In July 1879, jaundice appeared with white stools, deep-coloured urine. Later in the year appeared the biliary acids and leucin. Left India in 1880, and passed five weeks on the Continent without advantage. Suffered from recurrent congestion and enlargement of liver, with severe pain. During these attacks the liver enlarged up and down and increased in thickness. At such times the skin

discoloration increased; there was general hepatic pain, headache, and pain in the eyes; urine dark and stools dirty white. At times the intervals between these attacks were prolonged, and a fair approach to health was for a time made. She always suffered from general pruritus and sleeplessness. In July 1880 she was attacked with fever, pain over the right kidney and loins, and in the pit of the stomach. This pain became so severe that during a whole night she was kept under the influence of chloroform. She vomited a bright green fluid mixed with food. Had abdominal pain most pronounced over the caput coli. Temp. 100° Fahr. The attack was a severe one. Symptoms gradually subsided. Condition very unsatisfactory. On August 7 passed a naturally coloured evacuation, but this change for the better lasted only five days; then white stools reappeared, with hepatic uneasiness. And so the case lingered on, the liver often quiescent, at times passing bile in small quantity, at other times the motions were colourless. In July 1881, her condition was considered critical. Her pulse was 112, weak and small. The temperature 100°, and not only was there pronounced jaundice, but her legs began to swell, and assumed a state resembling phlegmasia dolens; and the cramps in the legs became so severe as to require the continued use of chloroform. Dr. George Harley's opinion was now obtained, and he at

once diagnosed the case as one of impacted gall-stone, and prescribed for her accordingly. There was pain over the popliteal, and pain with distinct hardness over the femoral veins. The strength became impaired, the skin discoloration increased, and the temperature over 100°, being higher in the evening. Pulse small, weak, and 112. On July 24 a free passage of bile ended the attack. No calculus was detected, nor did pain in the liver accompany or immediately precede the appearance of bile. For eleven nights good sleep was obtained without narcotics. In August there was a slight relapse with fever. Pulse 110, temp. 103°, liver enlarged and tender. Complained of weakness. State unsatisfactory; skin very dark; pruritus nearly constant; insomnia the rule; heart weak. In September the liver became enlarged and tender, with slight fever. Bile frequently appeared after mild hepatic pills. On December 4 bile appeared in the stools, then disappeared to reappear in quantity on December 15, since which date its outflow has been natural and unimpeded. The liver is now (February 27, 1882) normal in size and free from uneasiness; the skin is clearing, the urine is free from biliary discoloration; pruritus has disappeared, so have headaches; the strength has returned; appetite and digestion good, and the patient is putting on flesh rapidly. She pronounces herself to be quite well. Appetite is good and spirits excel-

lent. Is able to take long walks without any feeling of distress. Her secretions are all normal. The change from valetudinarianism to health took place on November 15, and from that date there has been a rapid restoration to normal health and strength.'

The next case I shall relate is that of a certain Lady N——, who, though not yet cured, is certainly on the high road to cure, for she walked into my study this morning—February 23, 1882—as briskly as if she ailed nothing. This patient's case is well known to several members of the profession both in this country and abroad, from one of our London physicians, who has great faith in the efficacy of mineral waters, having two years before I saw her sent her to both Carlsbad and Vichy, in the hope that their waters would rid her of her impacted gall-stones. When first summoned to the bedside of this patient, which was on February 27, 1878, she was suffering from a violent paroxysm of gall-stone colic. Her stools were pipeclay-coloured, her urine as dark as porter, and the colour of her skin an incongruously combined mixture of black, green, and yellow. Indeed, such was her strange complexion, that, as she subsequently told me, she could never go for a drive in the open carriage without a thick veil, as people stared at her so ; and when she walked in the streets the children would turn round and call out to each other to look at the 'funny woman.'

As it is over four years since I first saw her, and she had been ill with the same symptoms and signs for two years previously to that, the gall-stone must have now been firmly impacted in the common bile-duct for at least six years, notwithstanding which she is not only still alive, but in good health, and is even now passing small quantities of bile by stool, while the urine is at the same time only of a dark straw colour, and her complexion whitish-yellow. So well indeed is she that she was able to dance at a ball a few nights ago, to the amazement of all who knew how ill she had previously been, and how nearly she had approached the brink of the grave.

She has been treated by duct-dilators and biliary solvents at different times and in different ways. Alkaline carbonates have been freely used, as well as the iodide of potassium and bromide of ammonium, with occasional mercurials.

Great benefit was derived from benzoates, chiefly given in the following manner, which is a favourite formula of mine :—

℞ Acidi Benzoici . ʒ ij.
Potassæ Causticæ . ʒ ss. (*vel* Liq. P. ʒ ss.)
Aquæ Destil. . . ℥ vj. M.

Dissolved with heat, and a tablespoonful taken in water thrice a day.

The next case is related chiefly for the benefit

of the reader who, being but little versed in the clinical history of gall-stones, may perchance doubt not only the diagnosis but the benefits of the treatment in the foregoing cases, from the fact of no stones having even so much as been suspected of having been voided by the patients. Regarding the nature of the case now to be recorded, there cannot possibly exist a shadow of doubt, for not only one, but actually four stones were picked out of the fæces within the brief space of a month after the treatment began. The case has another advantage, for it had originally been mistaken for one of cancer, and, had a change of treatment not been promptly adopted after the error in diagnosis was discovered, would in all probability have terminated fatally—like most other cases of a similar kind. When I come to the chapter on cancer of the liver, I shall take occasion to point out the landmarks on which I rely in making a differential diagnosis in these cases. Meanwhile I shall give a bare statement of facts.

The case was that of a lady of some sixty summers who was sent from Devonshire, shortly after she had been seen in consultation (along with her usual medical attendant, Mr. Edwards) by Dr. Budd of the Exeter Hospital, who diagnosed the case as one of cancer of the liver. On her arrival in town I was asked to go at once to the hotel and see her. I found her much exhausted, from the effects no doubt

of the long railway journey. Her skin was not deeply jaundiced, having more of a greenish livid than of a yellow colour, not at all unlike what one sees when slight jaundice accompanies the cancerous cachexia. The tongue was foul, the pulse rapid, the skin hot. The liver moderately enlarged. Tender on pressure, smooth, and hard to the touch. The stools were pipeclay-coloured. The urine scanty, very dark in tint, and with a copious deposit of lithates. On careful palpation I detected a distended gall-bladder, which was acutely painful on pressure. She said she had never passed gall-stones. Nor had she at that time suffered from what could be put down as hepatic colic, though she admitted having occasionally suffered from stomach-ache. I at once diagnosed the case as one of gall-stone impacted in the common bile-duct, and proceeded to treat it accordingly. To make a long story short, I may skip over details until that day three weeks, when a gall-stone the size of a small hazel-nut was put into my hands. Two days later another, the size of a field bean, and four days subsequently a third, the size of a small hazel-nut, all three more or less circular, without a single facet on any of them. All of an exceedingly dark and rather roughish exterior, with a pale fawn-coloured crystalline interior. After the passage of the stones the patient got rapidly well, and I very soon had the pleasure of sending her back to Devon-

shire, cured of her 'cancer.' The day before she left, her maid made a voluntary confession, in somewhat the following words :—

'I got tired of searching the dirty stools for the stones, and I was just making up my mind to do so no more, when (five days before I gave you the first stone) I found in the middle of the stool what I thought was a boy's marble, so I picked it out and washed it. Then, seeing it was not a marble, I wiped it quite dry, and, in order to find out what it was, I tried to cut it in halves with a pair of scissors, when to my surprise it broke into a lot of shiny-looking pieces. I then thought it must be a gall-stone. And as I was afraid you would be angry at my breaking it, I made up my mind to say nothing about it and threw all the little bits into the fire, where they blazed away like wax. Although I said nothing about it to anyone, I now looked far more carefully in every stool, and on the fifth day I found the one I gave first to you. So mistress, I know, has at least passed four of them.'

There is an important moral to be drawn from this anecdote. For supposing this maid had possessed a less inquiring mind, or no scissors had been at hand, the true nature of the marble would never have been discovered by her, and not only would it most probably have been consigned to the w.c., but no more stools would have been searched for gall-

stones, and her mistress and I should have been *confidently* informed that no stone had ever passed. And, although the patient got well under the treatment, I should most probably never have had the credit of having either made a correct diagnosis, or been successful in getting rid of the gall-stones. I think the reader will cease, after reflecting on this case, to feel the slightest surprise at my having said that the longer I live the less importance do I attach to the non-discovery of gall-stones in patients' stools, and that too even after there is distinct symptomatic evidence of their being passed into the intestines.

The above case is an exceptionally favourable example of the benefits of energetic gall-stone treatment. It and the one I recorded at page 650, in which thirteen stones were passed, are two of the speediest cases of cure I ever had, and that is saying a great deal, seeing the immense number I have had to do with.

Now the sequel of the above case has yet to be told. The lady is still alive at the age of 74, has grown stout, and is, as far as the hepatic symptoms are concerned, comparatively speaking, well. She however suffers from other things—but neither from cancer nor gall-stones.

Treatment of Gall-stones seeking an Exit by Perforation of Tissues.

When we have reason to suspect that a gall-stone is working its way out of the gall-bladder or out of a bile-duct by ulceration, all our efforts must be directed to aiding that process, both by accelerating it and by mitigating its serious consequences.

Strict rest is to be enjoined, hot fomentations are to be assiduously applied to the tender locality. The strength of the patient is to be carefully supported by a judicious selection of easily digested non-stimulating animal foods, in the shape of nourishing soups and drinks. No solids, sweets, or fatty foods are admissible.

In cases where the gall-stone or stones have caused rupture of the gall-bladder or bile-duct, or where the ulceration has opened a blood-vessel or into the peritoneal cavity, all that can be recommended to be done is simply to use the best means which suggest themselves, under the circumstances, to ward off the cause of death. I cannot particularise any special line of treatment to be adopted in the manifold possible forms of complication which may arise in the erratic course of a gall-stone, but merely recommend the practitioner to remember that in all bad cases in a multitude of counsellors there is wisdom—provided the counsellors are of the right sort—and it is always good policy to have some one else to share the responsibility

when a suddenly fatal and unexpected termination may possibly occur. However, I think I may venture to give here at least one valuable piece of special advice in the

Treatment of Perforation with Hæmorrhage.

Which is, immediately on blood appearing in the vomit, the stools, or the urine, or even when invisible internal hæmorrhage is, on account of the symptoms, suspected to have occurred, no matter into what cavity or tissue, to APPLY ICE over the supposed seat of the rupture. I may as well incidentally remark that often the worst cases are those where no blood appears externally. And even in them a freezing mixture of ice and salt, if speedily had recourse to, is most useful. It being the most powerful of all styptics. Don't make the mistake, however, of putting the freezing mixture into a waterproof bag, and then applying its dry external surface to the patient's skin. For as waterproof cloth is a bad conductor of cold, it is absolutely essential to *wet the surface of the bag* which is to go next the patient's skin. The cold cannot be too intense. For in order to cope successfully with an internal hæmorrhage through the instrumentality of the external application, it is essential that it should be sufficient to freeze the external parts. A little blistering of the cuticle does no harm, as it produces no more dis-

agreeable effects than a case of cantharides blistering, and heals just as readily. The giving the patient lumps of ice to suck is an unimportant adjunct. For unless he swallows the fragments whole, the ice becomes warm water before it reaches the stomach. The next thing is to administer a potent astringent. Of these our Pharmacopœia possesses a superabundance. Lead and opium, gallic and sulphuric acids, krameria and kino, &c., &c. But there is a formula which has proved so successful in my hands that I have given it at page 266, in the hope that, either as it stands or with some modification, it may be found equally useful by others. A dose being given as frequently as the urgency of the symptoms demands. When there is fear of collapse, balsamic astringents ought to be given. Turpentine, though not one of the nicest, is one of the best.

In almost hopeless cases yet another plan may be tried, and that is the newly proposed one of injecting freshly-drawn warm blood into the peritoneal cavity. It is said not only to produce no disturbance, but soon to disappear by absorption.

Treatment of Gall-stones impacted in the Intestines.

It will, I think, be readily admitted that in the preceding pages it has been clearly shown not only that the evil results of a gall-stone do not always cease when it has reached the intestines, but that even

death itself has again and again resulted from the impaction of a gall-stone in the intestinal canal. When we have any suspicion that the stone is large, our treatment must be carefully continued until its extrusion by the mouth or rectum has been accomplished. The removal by operation of a gall-stone from the ileum in a case of acute intestinal obstruction is recorded by Mr. Bryant in vol. xii. of the Clinical Society's 'Transactions.' The patient, a married woman aged 50, had never had any illness of any kind until the commencement of this attack. Which began by her waking at night with severe abdominal pain and sickness. The vomiting being greenish led to the belief that it was simply a bilious attack. On the following day the vomited matters became fæcal, and the abdominal pain excruciating, especially towards the left of the umbilicus. The countenance was anxious, and the pulse feeble. As death seemed imminent on the third day of the symptoms of strangulation, the abdomen was opened from the umbilicus four inches downwards, and a hard ovoid body was detected in the lower part of the ileum, about a foot above the ileo-cæcal valve. The intestine being distended above, and collapsed below the seat of the obstruction. An incision was made into the gut, and a gall-stone measuring $1\frac{7}{8}$ inch in length, $1\frac{1}{8}$ in diameter, and $3\frac{1}{4}$ in circumference, weighing 238 grains, was extracted, and the wound immediately

afterwards stitched up with carbolised catgut. The patient never rallied, but died in eight hours. At the autopsy the wound in the intestine through which the calculus had been extracted was found well sealed up with new material. There was some blood-stained serum in the abdominal cavity, but no fæcal matter. The gall-bladder was absent and replaced by a thickened pouch, about the size of a walnut, and firmly adherent to the hepatic flexure of the duodenum, in which was a slight annular constriction at a point where it communicated by a small opening with the gall-bladder. The reason why she had never been jaundiced was, of course, that the stone had ulcerated its way directly from the gall-bladder into the duodenum, and never had the chance of blocking up the common bile-duct.

I may allude to another case in which Mr. Hugh R. Ker removed by abdominal section a gall-stone, as large as a pigeon's egg, from a patient suffering from intestinal obstruction of thirty-six hours' duration. The stone was situated immediately above the ileo-cæcal valve. The wound was carefully brought together by a catgut suture. Death took place four days after the operation, but no *post-mortem* examination was allowed.

Artificial Removal of Gall-stones from the Hepatic Appendages.

There is a certain class of cases where I would follow the advice of a celebrated French surgeon of the last century and unhesitatingly recommend the removal of gall-stones—when dangerously impacted in the common bile-duct—by the surgeon's knife. Although I shall have a good deal to say on this subject when I come to the consideration of diseases of the gall-bladder, I may here mention that I do not consider the removal of gall-stones from the living human body, though a bold, a hazardous operation. Indeed, I have but little doubt it will sooner or later come to be an operation frequently practised. For once the existence of the offending foreign body has been accurately ascertained, I see no reason why it should not be removed. In my opinion, judging from my experience in operations on the gall-bladders of animals, the operation of choleo-cystotomy is not even at the present moment one whit more dangerous than that of lithotomy, and ought, if proper precautions are taken, to be infinitely less so. I think, from the fact that not only the presence of gall-stones, but even their exact situation, when once they have become firmly impacted in a duct, is in general easily ascertained, that no hesitation need be felt in suitable cases early to call in the assistance of a surgeon.

In order to show that there are good grounds for

recommending the removal of gall-stones in suitable cases by making an artificial opening into the common bile-duct when they are giving rise to dangerous symptoms, I may refer to cases where it has been successful when done accidentally, as well as when done intentionally.

One day in speaking to Sir James Paget regarding the possibility of successfully establishing a biliary fistula in the human subject in cases of permanent obstruction to the entrance of bile into the duodenum, he related to me a case where he had seen a gall-stone extrude itself spontaneously from the gall-bladder through an inflammatory opening in the right hypochondriac region of a patient while he was being examined in his own study ; and as I naturally enough manifested great interest in the case, he kindly sent the patient to me a few days afterwards. The patient, a clergyman of over eighty years of age, when I examined him, had two fistulous openings in the abdominal walls just over the fundus of the gall-bladder. One of these was closed up, but from the other exuded a small quantity of bilious-looking pus. There was distinct tenderness on pressure over the seat of both of the fistulæ, and notwithstanding that the first fistula had formed nearly three years previously and discharged ever since, the gentleman, considering his great age, appeared to be in the enjoyment of excellent health. The history of the case as given to

me by the patient himself, in a letter dated 4th December, 1879, is the following :—

'The first detection of a formation on the right side was on March 15, 1877, closely succeeding a lengthened and severe illness from congested lungs. I had eminent physicians, who failed to find the enlargement, so I suppose it did not exist. From May 20 I was in Scotland walking as well as any of my age, and feeling no inconvenience and no pain. About the second week after my return from Scotland, it felt as if matter were forming, and on October 11 it came to a head and broke, discharging *two* gall-stones, about the size of a small pea, in the presence of Sir James Paget. No matter has ever passed. Nine or ten stones and crumbs of stone have come away. Their passage caused very little pain. The first orifice, after three or four months, healed, and a second opened. I do not think they have ever discharged together. The discharge never ceased.'

In the 'Lancet' of January 5, 1878, Mr. Cookson relates the case of a gentleman aged 65 who passed a number of calculi through a fistulous opening in the abdominal parietes. The history is briefly as follows :—

He complained of nausea and vomiting, with great pain at the junction of the right hypochondriac and epigastric regions, where there was a visible enlargement. For two years previously he had felt pain in this situation when leaning forward or against

anything. Over a spot which he said he could cover with his thumb, situated immediately below the ribs, and about three or four inches to the right of the median line.

At length an abscess formed, and burst about an inch above and a little to the right of the umbilicus. It discharged a quantity of fœtid matter along with from twenty to thirty gall-stones. Which were almost colourless, friable, and soluble in ether, varying from the size of a pin's head to that of a field pea. The largest weighed sixty grains. For several days two or three calculi came away daily. The discharge, which was at first thick and purulent, in a few days became thin and serous, but contained no bile. After this the patient's health rapidly improved. The nausea and vomiting ceased, and the pain and tenderness disappeared. Eight days later about a dozen more calculi were extracted by means of a probe, and the swelling and induration which up to this time had existed around the umbilicus now rapidly subsided. On the following day some more small calculi were discharged, and among them a large triangular one which passed with difficulty through the opening, which was of the diameter of a writing-quill. A probe was passed into it, and its depth was found to be four inches. For the first time the discharge was noticed to contain bile, and on pressure being applied over the liver two or three drachms of it flowed out

This is about the usual mode by which gall-stones make for themselves a direct exit through the abdominal walls ; but in further illustration I may briefly refer to the case published by Mr. Booth in the 'Lancet' of March 11, 1882.

He says that his patient, a lady aged 77, after having suffered from occasional attacks of biliary colic for ten years, had a painful swelling in the right side, which became red and burst, discharging a yellowish fluid. After remaining open for six months she observed 'a hard black point protruding from the orifice,' which hurt her on being touched. She dislodged it with a hair-pin, and found it to be a black pea-sized body. On the following day, two other similar concretions came away. Several more were afterwards extruded, and the colicky attacks ceased. For reports of other cases see page 629, and for further remarks on operative interference see what is said on the establishment of artificial biliary fistulæ in the chapter on gall-bladder diseases.

In parting from a patient who either has had a gall-stone, or is known to be predisposed to become the victim of one, it is always well to tell him not only to be careful about his diet and drink, but even about his mode of life. Of course he ought to be told to avoid fatty and rich saccharine foods, and all kinds of salted provisions, as well as every species of fermented drinks ; but in addition he ought to be told to

eat fresh lean meat and cooked vegetables ; to smoke little ; take plenty of muscular exercise ; live as much as is possible in the open air ; go to bed early and rise early, and never at any one time eat more than the wants of his system demand. In fact he ought to follow the advice of the Cid, which was 'not live to eat, but eat to live ;' for the pleasures of life greatly depend upon the 'liver,' as a superabundance of rich foods always, sooner or later, puts its functions out of order.

After having said so much on the treatment of gall-stone affections, it may perhaps be as well for me to remind the reader that success in practice in most instances depends less upon an intimacy with therapeutical formulæ than on a thorough acquaintance with the pathology and clinical history of the special case being prescribed for. Much on the same principle as the possession of a box of the finest paints does not of itself suffice to enable an artist to paint fine pictures. For, as the celebrated painter, Opie, told the young artist, who asked him what he mixed his colours with that enabled him to paint such beautiful pictures, that it was with Brains, in like manner the medical tyro may be told that in order to make therapeutical substances cure gall-stone diseases, they must, like the artist's colours, be compounded with 'brains.'

CHAPTER XII.

HINTS ON THE DIFFERENTIAL DIAGNOSIS OF HEPATIC FROM OTHER KINDS OF COLIC.

From its having been shown that gall-stone pain often exists without jaundice, it is easy to imagine how it may be confounded with other kinds of colic. The generic term of colic has been applied to every form of obscure pain occurring in the abdomen between the xiphoid cartilage and the umbilicus. The pain may have for its cause a gall-stone, a renal calculus, acute gastritis, enteritis, intestinal worms, invagination, strangulation, chronic copper and lead poisoning, ovaritis, or the inflammation of any other of the organs in that neighbourhood. Still all these forms of pain are included under the head of 'colic.' For no other reason, that I can see, than that the word 'colic' is derived from the Greek *koilia*, signifying the belly, and pain in the abdomen is, in homely language, a 'belly-ache.' But alas! this name, like the equally significant one of headache, is a term sometimes unwittingly applied to obscure and dangerous diseases. So that it is equally essential to the safety

of the patient and the reputation of the medical adviser, that the various kinds of colic should be correctly differentiated.

As I have already related a case showing how a hospital physician fell into the error of mistaking a case of acute gall-stone colic for a peritonitis supervening upon supposed perforation of the stomach, it may be as well for me to remark that mistakes of this kind are rare, and that it is the renal variety of colics alone which in general puzzle the practitioner. Indeed, the differential diagnostic characteristics of almost all varieties of colic are so well-known to every medical man that it is only the landmarks differentiating hepatic from renal colic that I now specially allude to; and in order that they may be engraven on the memory, I think I cannot do better than relate two typical cases illustrative of the difficulties encountered and of the means by which they may be overcome. In this instance, in order to cite cases possessing the advantage of being worthy of the name of crucial examples, I am reluctantly forced to select them from my own practice. However, when their histories are perused they will be found to be none the less convincing on that account. The first I shall cite is that of a middle-aged gentleman whose case was taken for an hepatic disorder by two eminent London consultants—one a physician, the other a surgeon—and by a third no less eminent consulting physician, as one of gastric derangement.

The second case was one which was mistaken for gall-stone by a general practitioner, and afterwards recognised as a doubtful example of that affection by a gentleman whose speciality is mental diseases. I need scarcely add that these errors in the diagnosis of the two cases led to forms of treatment not conducive to the welfare of the respective patients.

The first case fell under my notice in the latter part of 1871, when I was called to see a gentleman at Bayswater, in consultation with Mr. Riggall. After carefully examining the patient and hearing his history, I diagnosed the case to be one of renal calculus lodged in the pelvis of the right kidney. No sooner had I expressed this opinion than I noticed that it was received, both by the patient and his medical attendant, with a smile of apparent incredulity; and on enquiring why they doubted the diagnosis, I was told that the patient had already consulted two eminent physicians and a surgeon of reputed diagnostic skill in urinary affections, and that none of them thought that there was anything whatever the matter with the kidneys. Further, that while one of the physicians had diagnosed the case as one of 'gastric derangement,' the other, as well as the surgeon, had decided that it was a case of liver disease, and it was for the latter reason that I had been asked to see the patient. This was not a very pleasing piece of information. First, because my opinion was diametrically at variance with

the expressed views of three eminent consultants. Secondly, because one of them (the surgeon) was at that time thought to be a very high authority in urinary cases, and thirdly, I saw that I had both the medical attendant and the patient against me. Although, from the examination made and the data elicited, I did not doubt the correctness of my diagnosis, still the information I received made me pause and give due consideration to the different views Mr. Riggall told me that the other consultants had expressed. After attentively listening to each and all of them, as I still felt that there were substantial reasons for adhering to the opinion that the case was one of renal calculus in the pelvis of the right kidney, I put all the reasons in favour of renal colic categorically before Mr. Riggall, in order that he might act as umpire, and I soon had the satisfaction of bringing him round to my way of thinking.

The case was accordingly prescribed for as one of renal calculus, and it was arranged that I should again see the patient that day week.

On the day appointed Mr. Riggall met me at the door of the patient's house, and with a smile of satisfaction on his face presented me with a pill-box. On opening which I found the calculus, whose existence I had diagnosed the week before; it having been voided along with the urine that very morning. The result of the treatment having thus satisfactorily

proved the correctness of the diagnosis, I may at once proceed to give the history of the other equally typical case. Which, although a much longer time was required to verify the diagnosis by the result, in the end proved equally satisfactory to the patient and his doctor.

On February 15, 1881, a married gentleman, aged 27, was sent to me by Dr. Langdon Down, under whose treatment he had been for epilepsy, from his having in the course of consultation told Dr. Down that he suffered from, and had been for some time under treatment for, gall-stones, and the symptoms he described being anomalous, Dr. Down thought it advisable that I should examine him. I did so, and, taking his symptoms in conjunction with the result of an analysis of the urine, I came to the conclusion that he had *no gall-stone whatever* ; but a renal calculus, and that too of a mulberry nature. After this diagnosis was made, I was asked to take the case in hand. Accordingly I did so, and after nearly six months' weary treatment I had the satisfaction of getting the stone out of the pelvis of the kidney, and safely lodged in the urinary bladder. Seeing that the patient was an epileptic, and consequently it was most desirable that the stone should not be allowed to remain and set up irritation in the bladder longer than could be prevented, I sent him to Mr. Teevan to have it crushed ; but to my

chagrin, notwithstanding all Mr. Teevan's acknowledged skill, no stone could be detected. So the patient was returned to me. Being perfectly confident that a stone was actually in his bladder, and having learned by experience that the mere fact of a surgeon's not detecting a stone with a sound is no evidence whatever of the non-existence of a vesical calculus, whose presence in the bladder is invariably more easily diagnosed by medical symptoms than surgical manipulation, I at once put the patient on a course of treatment calculated to insure its speedy expulsion. The end soon justified the means ; for on September 22, 1881—that is to say about a month after the expelling treatment was begun, and 222 days from the time I first saw the patient—the patient called upon me with a mulberry *calculus* as big as a field bean in his waistcoat pocket instead of in his urinary bladder.

These two cases, I think, conclusively show not only how the symptoms of renal calculi may be mistaken for those of gall-stones, but how with proper care they may be differentiated, and likewise that when the diagnosis is correctly made treatment becomes, comparatively speaking, easy.

So thoroughly impressed am I with the truth of this latter statement, that year after year I used to tell the students attending my 'Urine Class' at University College, that the day is not far distant

when such a thing as a surgical operation for a urinary calculus will be regarded as an anomaly. For just as stones are now detected and crushed when small—being seldom or never allowed to become big enough to require the operation of lithotomy—so with our now improved methods of medical diagnosis (as they may always be detected when they are small enough to pass out of the bladder by the natural passage), we shall soon be able by our equally improving modes of treatment to insure their safe and easy expulsion from the bladder long before they become large enough to require to be broken into fragments by surgical instruments in order to admit of their being passed through the narrow urethral channel. It is an error to suppose that calculi must be smaller than garden peas in order to be able to pass spontaneously through the male urethra, for I have in my own collection numbers of calculi as big as field beans, and some even bigger, that have been expelled spontaneously by patients with very little and sometimes even with no pain whatever. The first knowledge the patient having had of their passage being their rattling into the chamber-pot. I have just paused to measure the dimensions of a calculus (which passed through the urethra of a gentleman who had never had so much as a catheter passed) given to me by Mr. Mason of Burton-on-Trent, and while its extreme breadth is exactly half an inch, its

extreme length is exactly three-quarters of an inch ! Yet it was voided with very little pain.

Having made this digression, as the treatment of calculi is a subject after my own heart, and every fragment of instruction I can give the reader will no doubt be acceptable to him, as the clinical history of urinary calculi is intimately associated with our subject, I will give the history of a case which will conclusively prove even to the most sceptical, that there are good and substantial grounds for my having said that the symptomatology of small calculi is a far more reliable diagnostic voucher of their presence in the bladder than the best metallic sound ever invented, even when under the guidance of the most experienced lithotritist, as well as prophesying that the days of operative procedure in the treatment of urinary calculi are rapidly drawing to a close. At least in all countries enjoying the advantages of possessing well-educated medical practitioners. This statement will, no doubt, only astonish those of my readers who are not versed in the last two centuries' chronological history of the treatment of stone in the bladder ; but for their sakes I shall relate a case the history of which amply justifies every one of the above assertions.

On a Sunday forenoon, in the month of November 1864, one of my out-patients at University College Hospital brought his son, aged 13 months, to my house, suffering from intense bladder irrita-

tion. The father and grandfather having both suffered from stone, and the visible signs manifested by the child (he could not yet talk, so no information could be obtained from him), coupled with that fact, clearly pointing to the existence of a vesical calculus; and as I make it a rule never to follow the reprehensible practice of prescribing for a stone without having first ascertained its true nature, when such a thing is possible, I determined to examine the child's urine before prescribing for him. But as the little fellow was either unwilling or unable to make water, I had no alternative but to draw some off. So taking a small 'infantile' silver catheter, I passed it into his bladder, and drew off as much water as I required. Then, before withdrawing the catheter, I made a search for the stone. Almost at once the end of the instrument touched it. Guessing that it was small, and could be very easily removed, at one sitting, by a lithotrite, and the child thereby instantly relieved of its suffering, I sent father and child along with a note to my then colleague, Sir Henry Thompson, with a request that he would kindly operate upon the patient. To my surprise, the father brought back the child to me, with a note from Sir Henry Thompson, saying that he had sounded the child's bladder, and that I was quite mistaken; for there was no stone whatever in it;

and, consequently, the symptoms must be due to something else. Having more confidence in the value of stone symptoms than in my colleague's infallibility, and feeling too that it was almost impossible for me to have mistaken something else for the peculiar sensation the contact of a stone communicates to a metallic catheter, I no sooner read the note than I ordered the man to replace the child on the sofa, and reintroduced the catheter. Again I detected the stone; and now, wishing to find out its size, I passed the forefinger of my left hand into the child's rectum, and guided the stone towards the point of the catheter. (In children, from the parts being small, and the tissues very thin, a great deal may not only be learned, but done, by manual manipulation.) Finding that I had the stone firmly between my finger and the point of the catheter, and the idea having struck me that I could possibly push the stone into the urethra by guiding it thither with the catheter, a trial was made, and the trial was successful. In less than three minutes I had the satisfaction of having the stone firmly lodged in the urethra, and within ten minutes more I had manipulated it —through the exterior walls always keeping it following the catheter—up the urethra to within halfway between the scrotum and point of the penis. Here it stuck, and I was just about prescribing a

powerful diuretic, in order to get the stone floated out, when it occurred to me that it might perhaps be as well to let my colleague not only see that physicians may detect calculi where surgeons fail, but kill two birds by one stone, and get him to cut it out with a knife from where it then was.

So I kept the child quiet on the sofa, while the father went back and asked Sir Henry to come round and bring a bistoury with him. In a few minutes he appeared. But he brought no bistoury, probably thinking that none would be required. I placed the little catheter in his hand and told him that if he passed it gently into the child's urethra, he would discover a stone. Before allowing him to do so, however, I secured the stone in its position by compressing the urethra immediately behind it with my finger and thumb, for fear it might accidentally slip back into the bladder on being touched with the point of the catheter, before he had time to satisfy himself of its existence.

As soon as he expressed himself satisfied that a stone was actually in the child's urethra, I handed him a bistoury, and requested him to cut down on it, and let it out. He did so, and the stone is now safe in my calculi collection—to me a not uninteresting souvenir.

As what has once happened may happen again, although it was no doubt by mere accidental good

luck that I succeeded in coaxing the stone into the orifice of the urethra, I am determined to repeat the experiment the first time I get the chance upon a child whose parts are sufficiently thin and lax to present even the faintest possibility of success. Even one or two failures, after having had such a success, would not, I think, deter me from repeating the trial ; for crushing even very small stones in infants' bladders is not always unattended with disagreeable consequences.

Now comes the all-important question : By what indications can a differential diagnosis be made between gall-stone and other forms of pain in the hepatic regions ? To give all the landmarks would simply be to write a treatise on the forms of pain in the stomach, diaphragm, duodenum, transverse colon, pancreas, kidneys, and all other organs and nerve-supplied tissues in proximity to the liver. Fortunately for me, however, such a course is unnecessary, seeing that I am addressing educated medical men who have already acquired a general knowledge of the subject. So all that is necessary on this occasion is to remind them of a few of the most salient signs and symptoms.

To begin with, it may be said that the chief guide to diagnosis lies in the previous history of the case, the organ that has been most prominently at fault, the condition of the patient which

led to the attack of pain, and the actual state of the hepatic, gastric, intestinal, renal, and other functions. When jaundice is present, the pain may in general be safely diagnosed as hepatic, and, if there be bloody urine, equally safely as renal.

I may remark that in those cases where there are no prominent signs or symptoms to indicate which organ is at fault, much can be learned by manipulation. Example: An excessive sensibility to touch all over the abdominal walls at once excludes the idea of the case being one of hepatic colic; for though patients cannot bear firm pressure as in lead colic, they can always bear gentle pressure and soft frictions with impunity all over the abdomen, except at one circumscribed spot. In peritonitis, enteritis, and gastritis, the tenderness is acute and diffused all over the epigastric region. In the case of renal calculus—no matter where the seat of pain may happen to be at the time of examination—it is always said to have begun in the back, like an ordinary lumbago; and, should the stone have entered the right ureter, and consequently the tender spot be in close proximity to the liver, the constant desire the patient has to make water may be the only sign we have to distinguish it from hepatic colic.

In the intestinal, renal, and hepatic forms of colic alike, the pain may be said to be usually paroxysmal; for, even in cases where there are no actual

intermissions, there are, in general, sufficiently well-marked remissions and exacerbations.

Moreover, in all these kinds of colic there is vomiting; but in the intestinal obstruction alone is the vomit stercoraceous, and in the hepatic distinctly bilious. The vomiting in renal cases, again, presents the peculiarity of occasionally continuing for weeks, not merely for days or hours, as in the other varieties of colic. I may here refer to a medical gentleman's case—a near neighbour of mine—in which the vomiting was not only incessant as in gall-stone, but continued for three weeks without a single day's intermission. Sir James Paget, when he saw the case along with me, suggested that, as the stomach was so irritable, it should be left entirely alone, and everything be given by the rectum. This was accordingly done, but without the slightest effect on the retching and vomiting, which went on for twenty-two days, when it all at once ceased on the stone's suddenly passing from the ureter into the bladder. In which viscus it was not allowed long to remain, for it was floated out from it into the chamber-pot in a day or two afterwards.

Rigors are equally characteristic of renal and hepatic colics, but are most severe in the latter. The pulse is usually described as weak and slow in both cases. Flint[1] says he has known it to be re-

[1] *Practice of Medicine*, 4th ed., p. 457.

duced to 20 beats per minute. This probably occurred when the patient was in a state of collapse. My experience is that little or no reliance, in a diagnostic point of view, can be placed on the condition of the pulse, for sometimes I have found it quick and strong as in inflammatory diseases, at other times weak and feeble. In exceptionally obscure cases, when, as it often happens, there is a doubt as to whether the pain be hepatic, renal, or intestinal, the way I usually set about my inquiries is as follows. Of course modifying the plan according to circumstances. To begin with, I ask if it is a first attack ; for if it is not, the history of the previous attack is almost sure to decide the nature of the case. The next question put is whether or not the previous attack was accompanied with or followed by symptoms of hepatic or renal disturbance. Particularly if there was either yellow skin or bloody urine. If the replies to these questions are in the negative, I then enquire into the possibility of lead or copper poisoning, and examine the gums for the metallic discoloration. If again the result is negative, and there are no visible signs to guide me, I at once turn my attention to the exact seat and quality of the pain. For I know that if it is hepatic it usually begins in the seat of the gall-bladder, and radiates over the right hypochondriac and epigastric regions towards the navel, and is, at the same time, accompanied with nausea and flatu-

lent distension. If, on the other hand, it is renal, the pain usually begins in the back, and travels downwards anteriorly in the course of the ureter. If the calculus is in the pelvis or ureter of the left kidney, there can be no, or at least but very trifling, difficulty in the matter. For the then position of the seat of pain is of itself sufficient to preclude its being mistaken for one of hepatic origin. In intestinal colic from chronic mineral poisoning the pain is not only less decidedly paroxysmal, but is located about the navel, and firm pressure at once reveals its true nature; for while firm pressure and rubbing are only tolerated, or at most only slightly comforting to the patient, in gall-stone and renal colics, they yield immediate and decided relief in all cases of intestinal colic, except those of the obstructive variety. Moreover, the previous history of a sallowness of the skin or a bilious personal or family history favours the idea of the case being hepatic. While the present or previous existence of blood, pus, or deposits of any kind, in the urine, is an equally probable sign of the case being renal.

Constipation is frequent in both hepatic and intestinal, scarcely noticeable in renal colics. Thick muddy urine is invariably present in renal, occasionally present in hepatic, but uncommon in intestinal colic. Old-ale-coloured urine is very common in hepatic and usually absent in renal and intestinal

colics. The metallic gum-line is a pathognomonic sign of lead and copper colics.

A retracted testicle is an equally pathognomonic sign that the case is one of renal colic. For while its absence is a negative sign of little value, its presence is a positive sign of undeniable fidelity. More than once I have founded what has in the end turned out to be a correct diagnosis on its presence alone. In order to guard against mistakes it is necessary not to forget the fact that a hepatic colic may occur in a patient with a renal history, a renal colic in one with a hepatic history, an intestinal colic in one with either, and *vice versâ*. It is further of service to remember, while attempting to make a differential diagnosis in the three different kinds of colic—hepatic, renal, and intestinal—that renal and hepatic colics are not unfrequently met with at different times, and even, though much more rarely, at the same time in the same patient. The uric acid diathesis is so often, so very often, associated with the cholesterin diathesis, that gall-stones and renal calculi frequently coexist in the same individual. It has occurred not merely once, but frequently, that I have had the same patient under my care for renal gravel at one time, and for hepatic calculi at another.

With the foregoing facts borne steadily in mind, coupled with a general knowledge of the most salient characteristics of the signs and symptoms occurring

in the other forms of disease likely to be encountered in the neighbourhood of the liver, but little difficulty will occur in the ordinary run of cases in coming to a correct differential diagnosis of colics. It must, however, be admitted that obscure cases will every now and again be met with which will tax the diagnostic acumen of the physician to its very utmost. As, for example, in rare cases like those cited at p. 633, where gall-stones have penetrated into the urinary organs, ulcerating their way into the pelvis of the kidney, then passing down the ureter, like a true renal calculus, and finally lodging in the patient's bladder, and there giving rise to all the ordinary signs and symptoms of a urinary stone. The difficulties of making a differential diagnosis in such a case are still further increased when the pain originally caused by the gall-stone has been situated (as in the case cited at p. 600) in the dorsal region, instead of in the neighbourhood of the gall-bladder. A knowledge of these facts, coupled with the finding that after distinct signs and symptoms of a gall-stone have come and gone, they are immediately followed by a series of signs and symptoms indicative of a calculus ulcerating its way into the urinary organs, and afterwards giving rise to equally definite indications of the presence of a foreign body in the urinary organs or their canals, will, I trust, aid the reader in his endeavours to arrive at a correct diagnosis in anomalous cases of renal and hepatic colics.

CHAPTER XIII.

CATARRHAL JAUNDICE.

This, the so-called Icterus Catarrhalis, originally of British nomenclature, may now be said to be a disease much more commonly spoken of in Germany than in England. Indeed, so great a favourite has the name become with German practitioners, that whenever they meet with a case of jaundice the pathology of which is to them unknown, they almost invariably dub it as an example of icterus catarrhalis. No matter whether it be a mere isolated sporadic case, or one of an epidemic multitude. We have recently been favoured by one of our cousins German with the history of an 'Epidemic of Catarrhal Jaundice.' Apparently so named for no other reason than that the author was in doubt regarding its true pathology, and, being in immediate want of a name for it, thought that 'Catarrhal Jaundice' would do quite as well for it as anything else. As there exists a great deal of ambiguity about the nature of icterus catarrhalis, it behoves me to give briefly as clear an explanation of its pathology as lies within my power.

I think I can make the pathology of it plain by comparing it with a pulmonary affection, and saying that it bears the same relationship to the jaundice of hepatitis as does bronchitis to pneumonia. That is to say, the bile-ducts in the one case, like the bronchial tubes in the other, are alone affected. While in hepatitis, as in pneumonia, it is the parenchyma, and not the mere ducts and tubes, that are implicated. And just as in bronchitis the tubes may become tumefied, and the passage of the air through them impeded, by a hyper-secretion of viscid mucus, so in icterus catarrhalis the bile-ducts may become tumefied, and the passage of the bile through them impeded in a precisely similar manner by a hyper-secretion of viscid mucus. Indeed, it has been asserted by some writers that they have found the common bile-duct completely plugged up by lumps of hardened mucus.

In the 'Deutsches Archiv für klinische Medicin,' 1875, Dr. Haenisch relates four cases in which he found the bile-ducts plugged up with mucus in an epidemic of relapsing fever with occasional jaundice which occurred at Greifswald. In all of these four cases, as there was also, however, more or less marked hepatitis, it is just as likely, indeed I think more likely, that the jaundice may have been the direct result of suppression from the effects of the hepatitis itself—as in other cases of inflamed liver—than due to obstruction the result of the plugging up of the

ducts by the mucus. For if jaundice is readily produced by a catarrh of the ducts, one would not unnaturally expect frequently to meet with mucus plugs at autopsies. While in reality exactly the reverse may be said to be the case.

However, I see in many cases no reason whatever to doubt the possibility of jaundice occurring as the result of viscid mucus plugging up the common bile-duct. For after the lucid description given of a mixed case of the kind by Dr. Hunter, referred to at p. 555, the only question of doubt remaining is as regards their relative frequency. Moreover, as we know that tumefaction of the common bile-duct is quite a common sequela to gastritis and duodenitis, from the inflammatory action extending itself from the intestines up the duct, and as both of these diseases, though not common, are far from rare, one is naturally enough led to believe that catarrhal jaundice must every now and again occur. I am unfortunately, however, unable to give any reliable signs or symptoms by which this peculiar form of jaundice is to be differentiated during the lifetime of the patient. For although we have several well-reported cases detailing the pathological conditions met with after death, no book or journal that I have consulted—and their number has not been insignificant—gives anything approaching to a clear and trustworthy description of its characteristic symptoms. So that I can say nothing further of

any clinical value upon the subject. Except it be as regards treatment, which is that Professor Gerhardt of Würzburg strongly recommends faradisation of the gall-bladder in so-called cases of icterus catarrhalis. He says that when the lower margin of the liver has been correctly ascertained, and the probable position of the gall-bladder made out, the electrode of a tolerably strong secondary current is to be pressed firmly against it, while the other electrode is placed in a parallel situation against the posterior abdominal wall. The current should, he says, be sufficiently strong to produce an audible contraction of the abdominal muscles, and be repeated again and again for a few seconds at a time. He moreover adds that he has seen during this treatment the specific gravity of the urine reduced from 1029 to 1023, and the colour of the secretion become much paler, the appetite at the same time improve, and the fæces become coloured with bile-pigment. ('Berliner klinische Wochenschrift,' July 6, 1873.)

As in Copland's Dictionary I find that reference is made to the treatment of jaundice by galvanism, the application of electricity to the liver in the manner just spoken of cannot be regarded as anything particularly novel. The only thing novel about it consists in the advantages which are said to immediately follow upon the application of the electric current to liver and gall-bladder. For my

own part I must add that although I can readily enough understand how a current of electricity passed into the nerves of the muscular fibres of the gall-bladder may induce the organ to contract and expel its bile—and consequently, were the obstructive body in the duct merely a mucus plug, the rush of bile along the tube very possibly might forcibly float it out—I cannot in the least degree understand how any current of galvanism, no matter however strong, could remove the inflammatory tumefaction and the other pathological anatomical changes which are said to exist in cases of catarrhal inflammation of the bile-ducts. For under no known conditions with which I am acquainted, can effused inflammatory products be removed, and their consequences be made to cease in the course of a few hours, by the application of a current of electricity through a diseased organ, such as the result of Professor Gerhardt's galvanic treatment appears to imply. Were, indeed, the removal of effused inflammatory products possible, we should in cases of catarrhal bronchitis have nothing whatever to do but pass a current of electricity through the patient's lungs in order to arrest the disease. Whereas, reasoning from analogy, it seems to me to be quite as unlikely that a catarrhal condition of the bile-ducts should be amenable to electricity as that a catarrhal condition of the bronchial tubes should

yield to the local application of galvanism. And inasmuch as scientific, or what is in general called Rational Medicine, is the mere application of 'common sense,' coupled with scientific knowledge, to medical matters, I think it would be well for us to remember what are the morbid anatomical conditions of the case ere we venture upon purely empirical forms of treatment. The least objectionable form of treatment, as far as I am aware, that has hitherto been proposed for this somewhat obscurely diagnosable form of jaundice, is the free administration of emetics, under the belief that they will not only tend to subdue the inflammatory condition of the bile-duct, but by their stimulating effect on the digestive canal increase the peristaltic action of the gall-bladder, and thereby favour the expulsion from the viscus of sufficient liquid bile to wash out into the intestines the mucus which by accumulating in the ducts had caused the obstruction.

Further information regarding the plugging of the bile-ducts by masses of hardened mucus will be found at pp. 704–5.

CHAPTER XIV.

JAUNDICE FROM POISONS.

Several organic toxic agents, such as the poison of snakes, fish, insects, and fungi, and all septic substances, as well as inorganic poisons, such as lead, copper, mercury, antimony, and phosphorus, produce jaundice; and, as it is still a disputed point whether these toxic agents act by suppressing or by obstructing the biliary function, I have purposely delayed their consideration until now, in order to introduce the subject here out of compliment to those who hold the latter view, though it be one to which I am diametrically opposed. However, it is in reality of very little importance in what part of a work of this kind the subject is treated, seeing that all theory is ephemeral, though fact is everlasting, and consequently the views on the *modus operandi* of poisons in producing jaundice propounded to-day may require material alteration when regarded in the as yet to us invisible pathological light of the morrow. (For effects of germ poisons, see pp. 333, 451.)

Virchow and his proselytes not only deny that the yellow discoloration of the skin which supervenes on the introduction of poisons into the humanbody is due to suppression, but even go so far as to assert that it arises from a physical obstruction to the outflow of bile into the intestines, caused either by mucus plugging up the bile-ducts, or by a tumefied condition of the ducts themselves. A view to which I take exception on what appear to me the following substantial grounds.

Firstly, the jaundice which supervenes in certain cases of poisoning is exactly like that following upon mental emotion in being exceedingly rapid in its onset. An hour or two sufficing to produce marked discoloration of the skin. Whereas, in all cases of jaundice from obstruction, one or two days are at the very earliest required for the development of a yellow cuticle.

Secondly, exactly as in cases of jaundice from suppression, the undoubted result of enervation, there is in all those the result of toxic agents a rapid development of cerebral disturbance, at the same time as there is in general an absence of the physical signs of liver-tissue derangement, which is never the case in any form whatever of jaundice from obstruction. Therefore my opinion is that toxic agents induce jaundice from suppression through the power they possess of paralysing the nerves which stimu-

late the liver-cells to secrete bile, just as some other poisons paralyse the nerves which call into play muscular action in the limbs.

Thirdly, the arguments of some in favour of the obstruction theory drawn from the occasional presence of bilious vomiting go for nothing, as bilious vomiting is even to be met with in cases of brain disease.[1]

Fourthly, the results of my own experiments on animals allowed to be bitten by poisonous snakes have shown that the bilious appearances of the stools are not due to the presence of bile, but of blood; and that extravasations are not only exceedingly common in the digestive canal, but under the skin, peritoneum, and even in the muscles themselves (see author's paper 'On the Action of Chemical Substances on the Blood,' in the 'Philosophical Transactions,' 1864–5, p. 687).

Fifthly, were we to adopt Virchow's views, we should at the same time be forced to accept an utterly untenable proposition, namely, the intervention of a congestive inflammatory condition of the bile-ducts. For mucus plugs do not block up normal ducts, and therefore, to produce the thickened mucus, we should be at the same time forced to suppose that there must have existed previous inflam-

[1] Hughlings Jackson stated (in a paper on 'Optic Neuritis,' read before the Ophthalmic Society, March 10, 1881) that in cases of optic neuritis the bile which is vomited often leads to the erroneous diagnosis of bilious or gastric fever.

matory action; and the existence of previous inflammatory action (unless it be supposed to have been limited entirely to the orifice of the common duct) presupposes hepatitis; while, moreover, as has been said, hepatitis is, as a rule, one of the pathological conditions which give rise to jaundice from suppression.

Sixthly, still further against this inflammation of the duct and obstructive mucus plug theory, stands the contradictory fact that jaundice occurs in dogs poisoned by phosphorus, even when there is a biliary fistula through which the bile could freely flow, if there were any to come away. In fact, the theory of obstruction, when philosophically considered under the lucid light thrown upon the mechanism of the whole biliary function by modern chemistry and experimental physiology, is untenable. For, as I assert, were we even to admit all the arguments that have been adduced in favour of the mechanism of the jaundice of toxic agents being obstructive, the one salient fact of the rapidity of its development in the majority of cases suffices to overthrow them all, seeing that it is absolutely impossible to imagine that any form of inflammatory action sufficient to bring about a yellow discoloration of the skin from an obstruction to the outflow of bile could occur in the course of an hour or two.

While in favour of the theory of suppression stands forth the salient fact that jaundice may arise as the result of enervation in a space of time so brief as to be measurable by minutes. I even dare to go a couple of steps further and boldly declare that our present knowledge authorises me to say that all the forms of jaundice arising from the introduction of poison germs, be they animal as in contagious jaundice, scarlet fever, typhus fever, and such like, or vegetable as in the numerous forms of malarial jaun dice, are due to suppression of the biliary function.

Nature does nothing on a small scale. All her actions, all her laws, are not only uniform in kind, but of universal applicability. The mountain and the molehill are not only built up on the same principles, but retained in their respective positions by the same great and universal law—the law of gravity. So it is in things medical. Notwithstanding, in some instances, their apparent anatomical dissimilarity, the pathological principles governing them are identical. I say, therefore, that if clinical teachers would only impress upon the minds of their pupils the necessity of applying to medical matters the same lofty philosophical principles which they see every day applied around them in the exact sciences, they would soon cease to look for causes in trifling abnormal individual acts, and be able to trace the origin of morbid effects in the outcome of great and

universally applicable organic laws. The due recognition and just comprehension of which readily enable the enlightened physician to understand most of the so-called obscure problems of medical science. It can scarcely be expected, however, in the present imperfect state of our pathological knowledge, that the application of abstract philosophic principles will remove every difficulty from the path of rational medicine; but, nevertheless, it may be safely opined that it will sweep many of them into oblivion. Even, for example, in the present instance, it shows us that the proximate cause of the jaundice resulting from the introduction of animal, vegetable, and mineral poisons into the human system, though it may be as obscure as midnight or as clear as noonday, in so far as its pathological mechanism is concerned, must of necessity be universally the same.

I shall attempt to give an illustration of the correctness of these views by here introducing a few words on the mechanism of the jaundice which occurs in these cases, as I think the generally accepted theory regarding it is quite erroneous, and that I can furnish a much better one.

The comprehension of the way in which toxic agents produce jaundice is, I imagine, simple enough, when viewed in the light of the facts I have already adduced, to prove that jaundice may arise from a suppression of the biliary secretion. For, as was

shown at page 105, the blood may become loaded and supercharged with bile-pigment when not a drop of bile is being secreted. While it has been equally well experimentally shown by Dr. Oskar Wyss ('Archiv der Heilkunde,' p. 469, 1867) that dogs poisoned by phosphorus become jaundiced, not only while their livers secrete no bile, but while the ducts themselves become filled up with their own normal white mucous secretion. An exact counterpart of the condition found in the human body referred to at page 105, in a case of undoubted jaundice from suppression of the biliary function. Where, in consequence of the liver ceasing to extract the biliverdin from the blood (and its formation out of red hæmatin going on uninterrupted), the elimination of it falls to the lot of the kidneys and skin, and, as a natural result, the urine becomes of a saffron tint, and the skin of a yellow hue (see page 308).

Although the above theories, like most other theories, are not entirely devoid of objection, I may add that, as it is always best to accept the lesser of two evils, I accept the theory that poisons produce jaundice by suppression, simply and solely because I cannot find a better. Even in some of what are called anomalous or exceptional cases, I think it ought to be readily admitted, if not easily understood, that there may be a suppression of the biliary secretion from the actual disorganisation of the hepatic

secreting cells induced by the specific effects of the poison on them. Indeed, it even appears that there are certain mineral poisons which induce jaundice from suppression by producing hepatitis. This is seen in some cases of phosphorus poisoning, in which, after death, is found a distinct inflammation of the liver. A fact that has been experimentally proved by Dr. Auspecht in the following wise. He injected a solution of phosphorised oil (one in eighty) into the subcutaneous tissue of the back of rabbits. Three milligrammes was the dose of phosphorus employed. Twenty-one animals were experimented upon ; and of these, thirteen died after one injection, two after two, three after three, and the rest after four, five, and nine. The conclusion arrived at by Dr. Auspecht is that phosphorus, or a modification of it, introduced into the blood, leads to a series of chemical changes in the liver-cells, with the formation of albuminoid granules and fat-grains in their protoplasm, though at first the liver-cells are not destroyed. If the phosphorus be administered too frequently, the albuminoid grains and fat-granules are no longer formed, but the cells become pale and glassy, with distinct nuclei, and the interstitial tissue becomes diseased. The changes observed are analogous to those which ensue in the kidney when the ureter is ligatured.

As additional collateral evidence in favour of the view that poisons produce jaundice by suppression,

either as the effect of enervation or hepatitis, I may mention that while experimenting in Paris in 1852 on the artificial production of diabetes in dogs by means of chloroform, ether, alcohol, and ammonia [1] introduced directly into the portal circulation, I noticed that the injection of some of these substances, more especially of chloroform, was occasionally followed by a discharge of dark, almost black, coloured urine, as in cases of paroxysmal hepatic hæmaturia, thereby showing that the extraction of the bile-pigment from the blood by the liver had been suspended by the mere introduction of the poison into its secreting cells through the direct instrumentality of the capillaries; and I think that this may be said to be an additional argument in favour of the theory that poisons induce jaundice by causing an arrest of the biliary secretion.

The following interesting case of jaundice supervening on phosphorus poisoning, with remarks thereon, occurring in the hospital practice of Dr. Habershon, is reported in the 50th volume of the 'Medico-Chirurgical Transactions,' p. 87.

The patient is described as a young healthy woman, aged 28, who took, it was supposed, about three grains of rat poison, mixed with water, on January 10. Burning sensation in the mouth and throat came on; the breath was phosphorescent;

[1] *Comptes Rendus de la Société de Biologie,* 1853.

vomiting and purging soon followed ; these primary symptoms subsided in two hours. For nearly forty-eight hours there was retention of urine. On the fifth day she was jaundiced. The abdomen was distended, and the liver much enlarged ; no pulse could be felt at the wrist. A small quantity of albuminous urine was passed after admission. The temperature of the body was very low. She rallied a little, but died suddenly nine hours later, after an attack of vomiting.

On inspection, numerous ecchymoses were found in the skin, mucous membranes, glands, and serous membranes. The brain was healthy. The liver was very fatty, its cells being distended with oleaginous globules. It weighed 79 ounces, that is to say twice as much as it ought to have done had it been healthy. The kidneys were pale. Even the voluntary muscular fibres were fattily degenerated, some fibres consisting of highly refracting granules ; the heart also was similarly fattily degenerated.

As may be noticed, though the phosphorus no doubt induced the jaundice, it could only have partially produced the fatty condition of the liver, as the organ was in such an advanced stage of fatty degeneration as to render it improbable that it could have been induced in so very short a space of time. The liver being said, though of the natural shape, to have been even tallowy to the touch, and under the microscope its cells found

quite full of oil-globules. The enormous quantity of fat present may be conjectured from the fact that its tissue actually, when a light was applied to it, burnt with a flame! The gall-bladder contained two drachms of bile, evidently not of new secretion, as it was dark-coloured, while newly secreted bile is of a pale yellowish-green tint. The presence of this small quantity of old bile in the gall-bladder still further supports my theory that all such cases of jaundice are the result of suppression of the biliary secretion.

At page 99 of the same 50th volume of the 'Medico-Chirurgical Transactions,' another case of jaundice brought on by acute phosphorus poisoning in a girl aged $4\frac{1}{2}$ years is recorded by Dr. Hillier. In this case the jaundice began on the fourth day, and death occurred on the sixth. The liver at the *post-mortem* examination was found 'very large, but excessively pale, as pale as chamois leather. . . . The hepatic cells were not easily made out, being replaced almost entirely by oil-globules.'

As regards treatment, all I need say is that as it is the cause, not the effect, that is to be treated in cases of jaundice from the introduction of poison into the system, the appropriate remedies for the special kind of poison inducing the jaundiced state of the system must be had recourse to, and in addition the bowels well cleared out, and the liver's action promoted by hot fomentations.

CHAPTER XV.

JAUNDICE FROM PERMANENT OBSTRUCTION.

The consideration of jaundice arising from a permanent obstruction to the flow of bile through the natural channels into the intestines is one of great importance to the medical attendant in a diagnostic point of view, as it invariably ends fatally, and consequently, if the case in the first instance fails to be correctly diagnosed, it not only to a certainty leads to a false prognosis, but at the same time also, in all probability, to grave errors in treatment, which in no case conduce either to the advantage of the patient or to the reputation of his medical adviser.

The subject of permanent jaundice naturally divides itself into two distinct parts. The one being the consideration of those cases arising from a congenital deficiency or a malformation of the bile-ducts, which will not detain us long. The other arising from an accidental occlusion of the hepatic or common bile-ducts in some part of their course. Which

will require minute consideration not only in consequence of the occlusion taking place under a very great variety of different circumstances and producing a well-marked chain of constitutional symptoms and visible signs, but from its actually being possible in many instances, by a judicious employment of therapeutical means, to very considerably prolong the life of the sufferer. And even when that can only be partially effected, it is at least invariably possible to considerably ameliorate his condition, and thereby soothe his passage to the tomb. From its being in this class of cases, too, that the advantages of scientific knowledge become most apparent, there are good reasons for my giving to the subject a considerable amount of attention.

Jaundice from Congenital Malformation of the Bile-ducts.

Probably some of my readers may think that I have already said enough on the subject of congenital malformation of the bile-ducts in Chapter VII., p. 295, while on intra-uterine jaundice. But as it seldom happens that cases of this kind are brought under the student's notice during his hospital career, and it is quite possible that a knowledge of its pathology may be turned by him to good professional account in practice, I think it will be well for me to say a few more words on the matter by relating the history of a case which was brought before the Patho-

logical Society in 1862 by Dr. Wilks. The child had never passed any meconium, the motions always being of a white colour. When a fortnight old, jaundice came on, and continued until death, at the age of six weeks. After death, the liver was found of a dark green colour, and, apparently, the gall-bladder was absent. On further examination, however, the cellular tissue, which appeared to occupy its place, was found to be traversed by a small canal, just large enough to contain a bristle; to this, however, no outlet could be found, and on endeavouring to discover the hepatic ducts, these, in like manner, could not be made out. The opening of the common bile-duct in the duodenum was natural, but no duct could be found to join it. It appeared, therefore, as if the larger ducts had become shrunken and entirely obliterated.

Through the kindness of Dr. Wilks I had the opportunity of making a microscopical examination of the liver. The hepatic cells, I found, were very small in size, much broken up; very few possessed nuclei, and all were deeply tinged with a brownish-yellow colouring matter. Scattered throughout the hepatic tissue, I found numbers of well-formed cholesterin crystals, like those represented in fig. 14, p. 551.

This case of itself shows the pathology of congenital obstruction of the common bile-duct so clearly

that I may, without further comment upon it, at once proceed to the consideration of

Jaundice from accidentally acquired Permanent Occlusion of the Bile-ducts.

As a knowledge of this subject is, I believe, as already said, of vast importance to the practitioner, in order to prevent his falling into the mistake often committed—too often indeed—of giving an erroneous prognosis, I desire that particular heed be given to the theories as well as facts which are put forward in this chapter.

In order to be able to follow my usual habit of attempting to kill two birds with one stone, and thereby avoid unnecessary repetition, I shall select as a typical illustration of this form of jaundice one of the most obscure and consequently most difficult cases to diagnose and treat of all the known forms of hepatic disease arising from a permanent obstruction to the exit of the bile from the liver into the intestines. Namely, that which follows the slow and insidious steps of a cicatrising duodenal ulceration at and around the orifice of the bile-duct. Not one out of twenty, I might even say out of a hundred, cases of which are ever correctly diagnosed during the lifetime of the patient, and very many of them are not even correctly interpreted after the morbid condition of the parts has been revealed by *post-mortem* examination.

The case which I select will not only give a typical example of the pathology of these affections, but conclusively show what important adjuncts physiological and pathological chemistry are to the other very slender means we possess of arriving at a correct diagnosis in such puzzling cases.

The case, moreover, is one of a permanent occlusion not only of the orifice of the common bile-duct, but of the orifice of the pancreatic duct as well, by a cicatrised ulcer. I say that this may be considered to be the most typical form of all the permanent jaundices arising from accidental obstruction, from the fact that, as the ulcer heals *very slowly*, the orifice of the bile-duct is equally slowly and gradually rendered impervious to the passage of bile into the intestines, so that the tinging of the skin is gradual. Actual jaundice never in these cases making its appearance until the orifice of the bile-duct has become completely occluded, and when the occlusion of the duct is completed the case is hopeless. Yet, strange to say, as will be seen in the sequel, it is not even then beyond the power of therapeutical art to alleviate the sufferings and prolong the life of the patient.

Pathology of Jaundice from Slow Obstruction.

In order to explain this completely I shall point out the different stages of the process as it occurs in

the most typical—although it be at one and the same time the rarest met with—of all the various forms of slow obstruction to the common bile-duct. That of the more ordinary forms having already been explained. As the process of occlusion of its outlet gradually goes on, the common bile-duct becomes more and more distended by the retained bile, till it at length attains an enormous size. The gall-bladder, being equally prevented from emptying itself, likewise becomes stretched and dilated, until it may at last become not only palpable to the touch, but much bigger than an infant's head, and by projecting forwards become even apparent to the eye through the abdominal walls. This was at one time observed to be the case in the patient whose liver and occluded ducts are represented in Plate I., p. 113.

The distension of the bile-ducts is not limited merely to those situated external to the liver, but also affects those within the substance of the organ itself; and to such an extent may this be the case, that, on making a section of a liver that has long had its common duct obstructed, a number of large excavations may be observed all over its divided surface. Which excavations are nothing more than the open mouths of the transverse sections of the dilated intra-hepatic bile-ducts. Such a state of matters is tolerably well represented in the exposed surface of the perpendicular section of the left lobe of

the liver in Plate I. Further, the effect of obstruction to the exit of the biliary secretion and its consequent accumulation, is not confined to the mere distension of the ducts, but causes various changes to occur in the parenchyma of the liver itself. The first stage of these tissue changes induces a general increase in the size of the organ, arising partly from the accumulation of the bile, and partly from the portal congestion caused by the pressure exerted on the vessels by the distended ducts. In the second place, gradually from the state of matters here described being continuous and progressive, the parenchyma of the organ becomes itself affected, partly from the direct pressure exercised upon it, and partly from the derangement of its nutrition produced by the interruption to the hepatic circulation. So that, after a time, the enlarged liver slowly and by degrees diminishes in dimensions, until it at length regains its natural size. Thereby rendering, at this period of the disease, when the bulk of the liver has become again normal, the diagnosis of the case extremely difficult. This state of matters is seldom, however, of long duration ; for, in consequence of the continued compression of the blood-vessels and parenchyma, the nutrition of the liver is so disordered as to lead to a gradual shrinking of its entire substance. In other words, to a general chronic atrophy of the organ.

It is thus seen how in *permanent occlusion* of the

common bile-duct the liver may be found *hypertrophied* in the *first*, of *normal dimensions* in the *second*, and *atrophied* in the *third* and *last stage* of the disease. Just as occurs in all other forms of chronic atrophy of the liver, from dram-drinking or any other cause whatever.

In cases of the kind here described, it is not at all unlikely that the enlargement of the liver in the earlier, as well as its atrophied condition in the later stages of the disease, may be mistaken for the cause of the jaundice, instead of the result of the mere arrest of the flow of bile. The first stage being mistaken for the ordinary enlargement of hepatitis. The last for that of the visceral atrophy arising therefrom. Thereby leading to grave errors in treatment. The clinical history of the case, taken together with a knowledge of the above facts, will, however, tend to facilitate the diagnosis. Thus, it must be ascertained :—

Firstly,—If the jaundice preceded the alteration in the size of the organ. This being a most important point in the diagnosis of jaundice from obstruction.

Secondly,—If there is an absence of any history of previous hepatitis, and the presence of any history of organic disease.

Thirdly,—If there is no evidence of any pulmonary or cardiac mischief likely to lead to passive congestion of the hepatic tissue.

Even with a knowledge of all these facts, how-

ever, it often baffles the skill and acumen of the ablest as well as the most experienced physician to discover the cause of the jaundice in a case of slowly progressing obstruction. Every now and then cases are met with, where the patient tells us that the jaundice has gradually come on without any assignable cause, and where, after the most careful and critical examination of his history, as well as of his physical condition, we fail to detect a clue to the diagnosis. Cases of this kind are far from uncommon, and this is the more to be regretted, seeing that unless we have a clear appreciation of the cause, it is not only difficult, but even dangerous to treat the symptom. For the injudicious administration of a remedy may here hasten the fatal termination we most desire to retard. The truth of this remark will, however, be better appreciated by the perusal of the chapter on the *rationale* of the treatment of jaundice. Meanwhile, it is here advisable for me to point out a method capable of yielding most important information, when all the ordinary means of diagnosis fail. I allude to the chemistry of the excretions.

CHAPTER XVI.

THE CHEMISTRY OF THE EXCRETIONS.

Although the pathological chemistry of the excretions is a branch of clinical study as yet in its infancy, from practical physiological chemistry not being a compulsory part of a general medical education, and few men having either the inclination or the means to devote two or three years to its study in the laboratory, it has, however, amply rewarded the few who have paid attention to it, for it may be truly said to have placed in the hands of the scientific physician a key to the detection of several diseases. And I trust to be able to show that even in the obscure cases of jaundice from permanent obstruction like the above alluded to, it not only gives a clue to their cause, but presents a guide to their treatment.

In jaundice arising from obstruction, the pipeclay stools are, as in the case of jaundice from suppression, entirely due to the absence of bile from the intestinal canal. The yellowness of the skin is in like manner

caused by the accumulation of the bile-pigment in the blood, from which it exudes into the cells of the *rete mucosum*, and in consequence of deeply staining them changes the complexion of the skin to a yellow hue. While saffron-coloured urine results in a similar way from the elimination of the abnormally accumulated bile-pigment from the blood by the kidneys. Instead, however, of these three conditions arising, as in the case of jaundice from suppression, from the arrest of the biliary functions allowing the constituents of the bile to accumulate in the circulation, in the first place, at least, they are the result of the re-absorption into the circulation of the normally excreted bile-pigment from the distended bile-ducts and gall-bladder. So that while, in jaundice from suppression, only those biliary products which like the pigment exist pre-formed in the blood accumulate in the circulation, in the first stage of the cases of jaundice from obstruction, the biliary products which are manufactured, like the bile acids, by the liver itself, equally with those which exist pre-formed in the blood and are only excreted by the liver, find their way back into the circulation, to be thence eliminated along with the urine. If, then, we can ascertain the presence or absence of these various products in the excretions, we are in a position to distinguish between jaundice resulting from suppression and jaundice arising from obstruction. Let us now see

what the chemistry of the excretions teaches us ; and to begin, we shall take the intestinal excretion.

Analysis of the Intestinal Excretion as an Aid to the Diagnosis of Obscure Cases of Jaundice.

Contrary to what is generally thought, the fæces do NOT consist of the effete products of our frames. Consequently their composition does not fluctuate (to such an extent as is generally believed) as the processes of animal life fluctuate.

The intestinal excretion in the natural state, indeed, only consists, firstly,—of those portions of our food which have resisted the action of the digestive juices ; secondly,—of the excess of the modified food remaining unabsorbed ; and, thirdly,—of the excess, as well as of the merely effete portions of the digestive secretions themselves. Consequently, under normal circumstances, the fæces vary more in consequence of the kinds of food taken than from anything else, and if from any cause the digestive secretions do not act properly on the food, the evacuation immediately becomes abnormal in character, and we are able to discover by analysis which of the digestive secretions is at fault. Thus, for example, knowing as we do that the saliva acts upon the starchy matters of our food, the gastric juice on the albuminoid, the pancreatic on the fatty, and that the biliary secretion so modifies the chyme as to allow of its rapid absorption by

the lacteal and portal vessels, we know further that if from any cause the elaboration or excretion of any one of these digestive juices is interfered with, more of the particular kind or kinds of food on which it acts, passes unchanged through the intestines. Thus, if the salivary secretion is affected, an unusual amount of unmodified starch is found in the stools.[1] If the gastric juice is defective, more albumen than is normal passes away unchanged in the stools, and so on with the others.

It is clear, then, that a chemical examination of the stools must afford important information regarding the presence, deficiency, or absence of the normal digestive secretions. A simple inspection of the stool will sometimes at once tell us whether or not bile is present. If it be present, the stool varies from a pale yellow to a dark olive-green hue, according to the kind and quantity of the biliary colouring matter present, and the nature of the food taken.

It must not be forgotten, however, that, unless care be observed, the colour deducible from highly-coloured food or the presence of blood may be mistaken for even an excess of bile. This remark is still more applicable to medicines ; for mercury, bismuth,

[1] A young woman aged 19, while a patient in University College Hospital, passed a solid, hard, white-lime-looking concretion, as large as a hen's egg, along with one of her motions, which I found by analysis was composed of agglutinated, hard, undigested starch. She had been in the habit of eating freely of rice-puddings.

iron, and some other mineral remedies, give rise to dark bilious-looking evacuations. So closely resembling bilious stools are these indeed in appearance, that the only way to distinguish them is by chemical analysis, when the presence of the mineral, together with the absence of the bile-pigment and the biliary acids (which, when properly looked for, are always to be found in normal evacuations), at once reveals the true nature of the case. I have frequently seen mistakes of this kind happen, and that too even in patients labouring under jaundice from obstruction. They having been thought to be passing the usual amount of bile in their stools, when in reality not a particle of bile was present. The dark colour being in these cases entirely due to the food and metallic remedies. Blood from the stomach or bowels is also not only apt to be, but actually is, constantly mistaken, even by medical men, for biliary matter. This is more especially the case when the blood has been, as in contagious jaundice and acute atrophy, acted upon by strongly acid intestinal secretions or gastric juice, which has the property of turning red blood brown. With these exceptions, the absence of bile from the stool is usually very easily ascertained by the naked eye alone. For if the patient is taking no highly-coloured food, or any of the medicines above indicated, and passing no blood, the fæces are of a dirty pipeclay colour. This tint not being due to the presence of

any new or foreign matter, but solely to the absence of bile-pigment. When bile is absent, the evacuations, besides being white, are usually of a most offensive odour. For, among other things, bile checks intestinal putrefaction and the development of offensive gases.

In addition to the colour and odour of the fæces, in cases of jaundice, another important indication is to be found in the presence of fat. The presence of fat in the stools was at one time looked upon as evidence of only pancreatic, at another time of only hepatic disease; now, however, experimental physiology has taught us, that it in some measure depends upon both. For while, on the one hand, the pancreatic secretion emulsions the fatty part of our diet, and thereby renders it capable of absorption, recent researches, as has been already pointed out, have established the fact that the biliary secretion also plays an important part in the absorption of the oleaginous constituents of our food. Bidder and Schmidt have shown that a dog, after ligature of the gall-duct, absorbs less than half the average normal quantity of fat; and by experiment it has been found that this arises from the circumstance that bile emulsions the acid fats, while pancreatic juice transforms the neutral as well as the acid oleaginous matters. The presence of fat in the stools may be due, then, partly to hepatic, partly to pancreatic derangement, but when much is present it is in-

variably due to both ; and I shall immediately point out how we can turn this fact to account in diagnosis, and discover, in cases of jaundice from obstruction, whether the seat of the obstruction is at the outlet or at some other part of the course of the common bile-duct.

Changes in the Kidneys and their Secretion.

Permanent jaundice arising from the occlusion of the common bile-duct occasionally gives rise to organic changes in the renal as well as in the hepatic organs, for the simple reason that the kidneys have to perform the vicarious function of eliminating the biliary products which normally pass from the body by the intestinal canal. This, in the course of time, leads not only to hypertrophy of the renal tissues, but, by keeping them in a state of permanent hyperæmia, in some cases to a condition of diffused local inflammation, which may end (as is seen in Plate II. p. 728) in the formation of numerous small renal abscesses. These abscesses may, however, have arisen not from hyperæmia, but from the plugging up of the capillaries by bile-pigment as explained at p. 732. So I shall not now make any further allusion to that point, but pass at once to the chemical examination of the renal secretion.

The urine affords us important information in all cases of hepatic disease. In fact, a naked-eye

Plate II.

R. Mintern Chromo lith.

Mintern Bros. imp.

Hypertrophied Kidney from a Case of Permanent Jaundice.

examination of the urine alone, in many cases, enables us to discover the presence or absence not only of jaundice, but of hepatic derangement before jaundice makes its appearance.

Diagnostic Value of the Colour of the Urine.

The urine of a jaundiced patient has invariably a peculiar tint, ranging from a saffron-yellow to a dark olive-green or almost black hue. It must not be forgotten that the colour of normal urine varies with its degree of concentration. Where little is passed, being of a dark, where much is passed, of a light colour; the depth of its hue depending on the degree of dilution of its normal colouring matter, urohæmatin. Again, it must also be remembered that there are many diseases which very materially alter the colour of urine, some only deepening, others actually changing the normal tint altogether, turning it from a straw into a blue or a green colour.

Foods, and medicines also, materially alter the colour of the renal secretion. Rhubarb, logwood, and santonin give to it a saffron hue, while carbolic acid, as well as arsenious acid gas, turns it of a black colour. Bearing in mind these facts, one should be careful before giving a decided opinion as to the presence or absence of icterus from a mere inspection of the urine. It is for this reason,

partly, that it is generally recommended in cases of suspected jaundice to pour a little of the urine on a white plate, and watch the play of colours produced by the action upon it of strong nitric acid. This method, however, is not always satisfactory, for the play of colours depends on the different stages of oxidation through which the pigment passes, and other animal pigments, besides biliverdin, unfortunately act in a somewhat similar manner. So, in order to insure greater exactitude, I always employ hydrochloric acid (which turns bile-pigment olive green) in testing urine for bile-pigment. Dr. W. Smith advises the tincture of iodine to be employed in the following way. Put an inch or so of urine into a test-tube, and slowly trickle a few drops of the tincture of iodine down the side of the obliquely-held tube till it reaches the urine, when, if bile be present, a beautiful bright emerald-green line is observed to form at the point of contact of the iodine and the urine.

In cases where it may be deemed advisable to ascertain the presence or absence of small quantities of bile-pigment, the spectroscope may be used for this purpose. Stokvis[1] recommends the urine to be treated with a small quantity of zinc chloride, and afterwards with an excess of ammonia. The mixture

[1] *Deut. Chem. Ges. Ber.* v. 583; *Journal of Chemical Society*, January 1873, p. 78.

becomes brownish-green when filtered or shaken with air. With the spectroscope it shows three characteristic absorption-bands, and a disappearance of the rest of the spectrum, commencing in the blue lines between *b* and *F′*.

A very simple and convenient way of testing the urine for bile-pigment without changing its physical characters is to separate it in combination with uric acid. This is readily done by simply acidulating the urine with a few drops of hydrochloric acid, and setting it aside for twenty-four hours to crystallise. The white uric acid in crystallising takes up the colouring matter, and assumes the hue of the pigment present in the urine. I have thus obtained crystals of all the different hues from a bright golden yellow tint through the intervening shades of red, brick-red, reddish-brown, blue, to a dark, almost black colour. This experiment has another advantage; for if we take a measured quantity of urine, and collect, dry, and weigh the uric acid obtained from it, we can readily calculate the total quantity passed in the twenty-four hours, and thereby be aided in diagnosing the presence or absence of malignant disease of the liver, as I shall afterwards have occasion to point out. As a rule, the urine of jaundice spontaneously yields copious red urates, and crystals of uric acid on simply becoming cold.

The urine of jaundice is generally described as being of a saffron colour ; but if I may be allowed to offer an opinion from my own observations, which are exceedingly numerous, I should say that in colour it much more frequently resembles old ale than anything else with which I am acquainted. On standing the colour changes very considerably, in consequence of the pigment becoming slowly oxidised by exposure to air.

The cause of the various tints of the bile-pigment met with in the urine of jaundice is easily explained on the theory of animal pigment oxidation in general, which I have fully expounded in my book on the urine, the sixth chapter being specially devoted to urine-pigments—yellow, green, blue, and black—so I need not take up the time of the reader by going into the subject here, further than to remark that the presence of blue or green urine in a case of jaundice has nothing whatever essentially specific about it, but is in reality a merely accidental concomitant of the disease.

When there is a very great excess of bile-pigment present in the blood, the kidneys have a difficulty in eliminating it. Occasionally it even chokes up the renal capillaries and tubuli uriniferi, and thereby complicates the jaundice by inducing secondary degenerative disease in the kidney. In such cases the external surface of the kidney, after the

removal of the capsule, may look as if it had been sprinkled over with ink, the black specks varying in size from the minutest visible point to that of a large pin's head. The chromo-lithograph, Plate II., represents a kidney in this condition, which was taken from the case of permanent jaundice from obstruction of the orifice of the common bile-duct, in a gentleman aged over 50 years, referred to at p. 769. As will also be observed, the kidney is studded over with a number of small abscesses, which I believe resulted from the blocking-up of the capillaries just alluded to by the bile-pigment. In the case in question no albumen was detected in the urine during life, and it was only on careful analysis, after the *post-mortem* had revealed the above state of matters, that a small quantity was discovered in the urine removed from the patient's bladder at the autopsy; and even then, had not the experiment been carefully performed, the presence of albumen would probably have been overlooked.

Diagnostic Value of the Presence of the Bile-Acids in the Urine.

All acquainted with the literature of jaundice know how hard a battle has been fought between two sets of observers in Germany regarding the presence of bile-acids in urine. One class, with Frerichs and Städler at their head, believe that the

biliary acids are decomposed in the blood, and are consequently never to be detected in the urine. The other class, headed by Kühne, state as positively that they have frequently detected these substances in the urine. Indeed, Kühne states that by adopting Hoppe's method[1] he never fails to detect the presence of the biliary acids in the urine of patients labouring under icterus, as well as in the urine of dogs with the common bile-duct ligatured. When first studying this question, I was very much perplexed by these contradictory statements, for neither the judgment nor the power of observation of either the one or the other of the disputants could for a moment be called in question; and on experimenting for myself, so unsatisfactory were the results at first obtained, that I almost threw the question aside in despair. On one occasion, however, I at length met with such unmistakable evidence of the presence of bile-acids in the urine in a case of jaundice, that I could no longer doubt the fact of their existence, and therefore was forced to search for an explanation of the previous contradictory results. Fortunately, it was not very

[1] The urine is boiled with an excess of milk of lime for about half an hour, and filtered to free it from the precipitate thus formed. The filtrate is evaporated to dryness, decomposed with hydrochloric acid, washed with water, and then extracted with alcohol. The alcoholic extract contains the bile-acids, which are recognised by Pettenkofer's test of sulphuric acid and sugar solution.

long before a solution of the difficulty was obtained, and, what was of still greater importance, led to the discovery that the contradictory results arose from a circumstance which might be turned to account as a means of differential diagnosis. The discovery simply was, that in certain cases of jaundice not a trace of the biliary acids is to be detected in the urine, although the bile-pigment may be present in abundance; while in certain other cases both biliary acids and bile-pigment occur in notable quantity. What, then, was the cause of this difference? Merely this. In jaundice from suppression the liver *does not secrete* bile; consequently, no bile-acids being formed by the liver, none can enter the circulation. They are therefore not to be detected in the urine. Only the bile-pigment, which is formed in the blood, is to be found there. In jaundice from obstruction, on the other hand, *bile is secreted* by the liver, and absorbed into the blood; and the bile-acids not being all transformed in the circulation, as Frerichs and his followers suppose, a portion of them is eliminated by the kidneys, and appears in the urine, where it can be detected by Hoppe's, Neukommen's, or Hilyer's method.

There are many analytical methods by which the presence of bile-acids in the urine can readily enough be detected by any one versed in physiological chemical manipulations. The simplest of

them at present is, in my opinion, that described by Hilyer, which consists in precipitating the bile-acids from the urine with an ammoniacal basic acetate of lead solution ; drying the precipitate, and extracting with hot absolute alcohol ; adding carbonate of soda, again separating with hot alcohol, and precipitating them from their alcoholic solution by pure sulphuric ether. This is only a rough sketch of the method, in order to give the reader an idea of it ; but for practical purposes he must consult the original paper, which will be found in the 'Chemical Society's Journal,' 1876, p. 445.

Alas, however, of what use is it to talk about the mode of chemically detecting bile-acids in a treatise like this, which is intended for the use of practical medical men ? Not one in a thousand of whom has ever had the opportunity afforded him of so much as even seeing bile-acids chemically detected in any organic mixture whatever, far less of having been taught how to find them in urine. Unfortunately analytical chemistry is a branch of knowledge which cannot be acquired in a day. For there is no royal road to its acquirement, and, as I said in the introductory chapter, he who desires to turn physiological chemistry to useful account at the bedside must be content to undergo the drudgery of learning it by working at the analytical table of a laboratory. For assuredly he cannot learn it in

the wards of a hospital. Scientific chemists in all countries, and at all times, have been most anxious to put into the hands of physicians a simple method of detecting bile-acids. But as yet, like myself, with the supposed simple test I proposed for them in my book on Jaundice twenty years ago, all have failed. The last simple method is that proposed by Strassburg. It is the modification of Neukommen's test (Pflüger's 'Archiv für Physiologie,' vol. iv. p. 461), and is the following. A piece of white filtering paper is dipped into the suspected urine, in which some cane-sugar has been previously dissolved. The paper is then dried, and, when dry, a single drop of pure sulphuric acid is allowed to spread rapidly over its surface. If bile-acid (glycocholic) be present, a mauve purple colour makes its appearance. If the filter paper be not dry before the sulphuric acid is added, the presence of water allows the sulphuric acid to char the sugared paper, and thereby mar the delicacy of the test.

Although I give this test as a rough and ready one for trial at the bedside, I cannot say I put trust in it, knowing by experience that, unless the purple reaction be exceedingly well-marked, it cannot be accepted as offering conclusive evidence of the presence of bile-acids. For in certain stages of oxidation, I find that bile-pigment, as well as the urohæmatin, which is constantly in greater or lesser quantity

in urine, gives an almost identical coloration. So that, in all cases of doubt, a chemical analysis must be had recourse to, before one can be certain either of the presence or of the absence of bile-acids.

As numbers of cases of jaundice result from suppression of the hepatic function, and as many of the cases of obstruction ultimately merge into the former, it is easily understood how the existence of the biliary acids in the urine has been so stoutly denied by some. A thousand negative, however, are of much less import than one single positive observation in deciding a question of this kind. And so long as there exists *one single* indisputably authentic correct observation, the opponents to it simply waste their time in attempting to dethrone it by piling negatives upon negatives. Even in true cases of jaundice from obstruction, bile-acids may fail to be detected, simply because there are none to detect. And why ? For the reason that as all chronic cases of jaundice from obstruction gradually, as I shall presently point out, merge, in consequence of tissue disorganisation, into jaundice from suppression, the biliary acids slowly and gradually diminish in the urine, until at length, for some time before the death of the patient, they entirely disappear. Although I called attention to this fact twenty years ago in my book on Jaundice, it was last year brought forward in Germany as a new discovery.

It will be remembered that, in speaking of the

bile-acids in the physiological chapter, I mentioned that while glycocholic acid is a crystalline, taurocholic acid is a non-crystalline substance. Tyrosin and leucin stand in a precisely similar relation to each other; tyrosin being a crystallisable, leucin a non-crystallisable substance. Now, taking this fact into account, together with the fact that when the bile-acids are allowed slowly to enter the circulation, they reappear in the urine, accompanied with tyrosin and leucin, and also with the third fact of these latter substances being found in the liver when the biliary function is interfered with, I am inclined to look upon tyrosin and leucin as products in some way or another connected either with the arrested or with the retrograde metamorphosis of glycocholic and taurocholic acids. Moreover, in one case I found, after injecting the bile of a healthy dog into the cellular tissue of another dog, crystals of tyrosin spontaneously form in the bile taken from the animal's gall-bladder after death, when the bile had merely been allowed slowly to evaporate. This result strengthens the foregoing opinion.

Frerichs states that he has never detected the biliary acids in the blood, even after bile had been injected into the circulation. Which may have arisen in his case, as it has done in that of some other experimenters, from a defective process having been employed for their detection.

In a remarkable case where one ounce of ox-bile, injected all at once, killed a dog in less than five minutes from the time it began to be introduced into the jugular vein, I detected the bile-acids in a clear extract of the blood with facility. This leads me to mention that, contrary to the statement of Frerichs, and in accordance with that of Kühne, the injection of pure bile-acids into the blood is very dangerous, and that even the injection of pure bile in small quantity into the cellular tissue sometimes proves fatal in the course of twenty-four hours, thereby showing that some of the constituents of the bile are highly poisonous. These I believe to be the bile-acids.

In illustration of these facts I may cite the following experiments :—

Into the cellular tissue of the back of a full-grown and healthy-looking English terrier dog, I injected the bile taken from the gall-bladders of three healthy dogs, two of which had just been killed, the other having been dead three hours. The bile was in the first two cases neutral, in the third faintly alkaline. All the three biles seemed perfectly normal. They contained no crystals of any kind. Eighteen hours after the operation the animal appeared quite well, and took his food heartily. Four hours later a remarkable change suddenly took place. The dog became dull and drowsy, and could not sustain himself on his legs ; when left to himself, he

lay on his side, and made not the slightest movement. He was not only paralysed, but even the nerves of sensation had ceased to act, for when his tail and feet were forcibly pinched, he appeared to be quite insensible to the pain. The pupils were dilated, and the body felt cold. Death occurred twenty-three hours after the operation. Urine and fæces were passed in small quantity immediately before death. The urine was strongly alkaline, and effervesced on the addition of sulphuric acid, thereby showing that it contained alkaline carbonates. Prismatic phosphatic crystals were present in the still fresh urine.

When tested for bile-acids, only the faintest trace was obtained, after the urine had been cleared with the acetate of lead and sulphuretted hydrogen.

The subcutaneous tissues of the abdomen and thorax were œdematous, but, within an hour after death, had not the disagreeable odour usually found in animals killed by the injection of pure bile-acids.

This experiment was again repeated with alkaline bile. Two ounces of ox-bile of a specific gravity of 1025 were injected under the skin of a large pointer dog. In twenty-four hours the animal was dead; the subcutaneous tissue all round the seat of the injection was red, inflamed, and infiltrated with blood. The urinary bladder was quite empty. The gall-bladder contained 1½ ounces of dark bile of a specific gravity of 1040. When examined with the micro-

scope, the blood was found to contain a large excess of white corpuscles.

This reminds me of a fact that I have omitted to mention—namely, that in a case of severe jaundice from suppression in a man aged 54, in consequence of cirrhosis of the liver, I found that the blood possessed a very treacle-like aspect. The serum was of a dingy yellow hue, and felt sticky to the fingers. Under the microscope the blood-corpuscles were found to be large and flabby, with a great tendency to adhere together by the edges, and become flattened on the sides wherever they came in contact. Moreover, the corpuscles looked as if they had no distinct cell-wall ; some, and that too in the freshly drawn blood, gave off buds, others actually split into two distinct corpuscles, each corpuscle, when separate, looking like a distinct red blood-cell. In fact, the blood looked more as if it had been acted upon by some powerful chemical agent than anything else. I again examined it after the death of the patient, and found it presented all the above characters in a still more marked degree. To the naked eye it had a viscid, tarry appearance.

Mr. Graham Brown, in 1875, read a paper to the Royal Society of Edinburgh, in which he stated that on injecting under the skin a sufficient quantity of the bile-acids to kill a rabbit in three or four days, severe diarrhœa and drowsiness, ending in somno-

lence, was the result. The acids reappeared in the urine; but there was no bile-pigment eliminated. The white blood-corpuscles appeared to be increased, and the red cells proportionally diminished, just as happens when the bile-acids are injected into the veins directly.

In one case I tried to imitate more closely the effects of natural disease by injecting slowly the bile of three healthy dogs under the skin of a fourth. The immediate effects of the operation itself being almost *nil*, and the bile being at the same time placed in a position favourable for its slow absorption—just as occurs in disease in the human subject—during the first two days after the operation the animal remained comparatively well. The urine was normal in appearance, and contained neither bile-pigment nor bile-acids. But on the third day the animal became ill, on the fourth jaundice set in, and on the fifth he died. After death the urine was found to contain not only bile-pigment and bile-acids, but also the diseased products, leucin and tyrosin; and, what was more interesting still, the urine was loaded with sugar.

These results strongly militate against the common theory of the bile being reabsorbed, in an unchanged state, into the circulation, after the completion of the digestive process.

It may be observed that I have taken no notice whatever of Professor Frerichs' theory regarding the

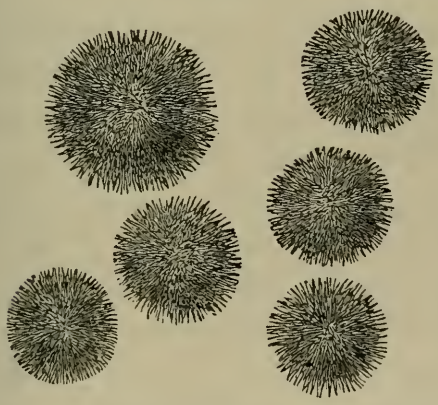
bile-acids being changed into bile-pigment. I have done so advisedly, feeling, as I do, that when that observer investigates the subject more fully, he will himself abandon so untenable a doctrine, founded, as it is, on an entirely erroneous notion regarding the nature and chemical composition of bile-pigment. The mere fact of bile-pigment being an albuminoid substance, and containing iron as one of its inseparable constituents, is sufficient of itself to show the fallacy of the theory of its being a product of a substance like bile-acid, which contains no iron and is not even so much as an albuminoid. Added to this fact the colour induced by sulphuric acid on the acids of the bile is as different in its chemical nature from that of an albuminoid pigment as any two substances can possibly be. They have no bond of connection whatever, except the mere trifling one of tint. All animal pigments, whether they be green, like bile-colouring matter, or red, like hæmatin, contain iron, and spring from the same source. Besides this, the mere fact of an increase of animal pigment being found in the urine after the bile-acids have been injected into the circulation, to which Frerichs attaches such importance, in reality proves nothing more, as Kühne pointed out, than that an increased destruction of blood-corpuscles has taken place. I have found the urine of dogs loaded with dark colouring matter after injecting chloroform and other stimulants into

their portal veins in order to establish artificial diabetes, or making them breathe or swallow carbolic acid, in which cases assuredly the presence or absence of bile-acids in the blood had nothing to do with the result.

Diagnostic Value of the Presence of Tyrosin and Leucin in Urine.

Tyrosin and leucin are two other abnormal products occasionally met with in the urine of jaundice.

FIG. 15.



Spiculated balls of tyrosin from the urine of a case of acute atrophy of the liver. When they were re-dissolved, purified, and re-crystallised, I found that they assumed the form of needles and stellate crystals, as represented in fig. 16.

These substances, which are supposed to be normal products of the pancreas, but not of the liver, although for many years known to chemists, attracted comparatively little attention until Frerichs discovered their diagnostic value in hepatic disease.

In cases of marked atrophy of the liver, the urine invariably contains tyrosin as well as leucin. When tyrosin and leucin are present in quantity, they are readily detected, all that is required being slowly to evaporate an ounce or two of urine to the consistency of syrup, put it aside during a few hours to crystallise, and then examine it with the microscope. The tyrosin is recognised by appearing in the form of spiculated balls not unlike a rolled-up hedgehog, with the bristles sticking out in all directions (fig. 15).

The following is an analysis of the urine in the case of acute atrophy occurring in a married woman, aged 17, who died of the disease in the third month of pregnancy, in which both tyrosin and leucin occurred, related at page 403 :—

Specific gravity	1028
Reaction	faintly acid (?)
In 1000 parts yielded:	
Water	948·860
Solids	51·138
Urea	30·000
Uric acid	0·375
Resin, mucus, and fat Biliverdin Urohæmatin Leucin and tyrosin	14·575
Inorganic salts	6·188

The urine in this instance was of a bright yellow ochre colour, and on simple evaporation yielded the crystals of tyrosin and leucin. The latter in very considerable quantity, but the former only sparingly.

The normal animal resin was markedly increased, and fats were present in unusual quantity. The nature of normal urine resin I explained in a paper on Urohæmatin in the 'Verhandl. der Phys.-Med. Gesellschaft zu Würzburg,' Bd. v. 1854.

The above analysis, when calculated on the twenty-four hours' urine, gives the following result :—

Water		372·000
Solids	organic	16·740
	inorganic	2·298
Urea		11·160
Uric acid		0·139
Resin, mucus, and fat Colouring matters Leucin and tyrosin		5·441

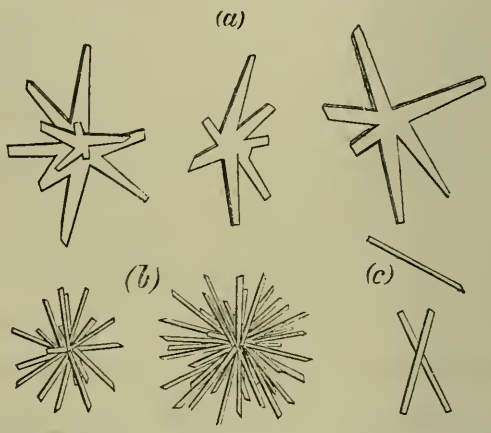
Tyrosin may be obtained in a perfectly pure state from the urine by adding to it a solution of acetate of lead till a precipitate ceases to form ; filtering and freeing the liquid from the excess of lead by a current of sulphuretted hydrogen; again filtering, and evaporating the clear solution and setting it aside to crystallise.

As I showed in my book on Jaundice (page 64), when the hedgehog-like spiculated balls of tyrosin are separated from the urine, and, after being purified, are allowed to crystallise slowly, they assume the form of beautiful white needles and groups of stellate crystals as represented in fig. 16.

Tyrosin can be readily recognised by putting a few crystals on a platinum spatula, adding a drop or

two of nitric acid, and evaporating to dryness. If tyrosin be present, the yellow residue thus obtained assumes a pumpkin hue on the addition of potash, and leaves on incineration a dark greasy stain. Frerichs recommends another test, namely, the following :—Put the suspected substance into a watch-glass along with some sulphuric acid, and after they

FIG. 16.



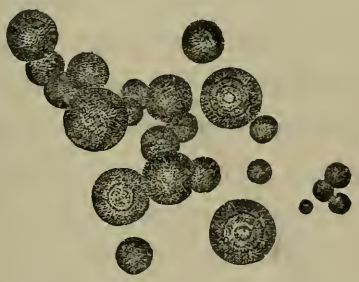
Crystals of pure tyrosin, obtained from the urine of a case of chronic atrophy of the liver following upon obstruction of the bile-duct. (*a*) Large crystals. (*b*) The more common form of the stellate groups of needle-shaped crystals. (*c*) Separate fragments of needle-shaped crystals.

have been in contact about half an hour, dilute the mixture with water. Next boil and then neutralise with carbonate of lime. Filter, and to the clean filtrate add a few drops of perchloride of iron, devoid of free acid. The formation of a dark violet blue colour indicates the presence of tyrosin.

Leucin again is known by its flat, circular, oily-

looking discs, without any crystalline structure. At the first glance, a globule of leucin might be mistaken for an oil-globule, not only on account of its microscopical characters, but also on account of its being lighter than water. The globules of leucin are, however, distinguished from those of oil by their being insoluble in ether. Besides this, the discs are occasionally opaque and laminated something like the granules of potato starch. They are then not at all

FIG. 17.



Dark globules of leucin of various sizes, resembling in appearance both globules of carbonate of lime and microscopic gall-stones.

unlike microscopic crystals of the carbonate of lime, or microscopic gall-stones ; but both the carbonate of lime crystals and microscopic gall-stones sink in water. The carbonate of lime is further differentiated from gall-stones by effervescing with nitric acid. (See case at page 584.)

The deposits of tyrosin and leucin are usually deeply impregnated with the colouring matter of the urine.

Since Frerichs' views were first published I have

found tyrosin and leucin in the urine both of cases of red, or subacute, and of the ordinary forms of chronic, as well as of acute atrophy of the liver. So that their presence may be regarded as an aid to the diagnosis of all kinds of hepatic atrophy. Although I must at the same time admit that their presence is more especially applicable to the diagnosis of the acute and subacute varieties of the disease.

The prophecy I made, in my book on Jaundice, in 1863, that future research would no doubt reveal other conditions of the liver, besides those of mere atrophy of the organ, in which both tyrosin and leucin would be found in the urine, has, it even seems, been more than verified by both continental and British physicians. For now it is found that both of these substances are to be met with not alone in what may be strictly called liver cases, but in the urine of patients suffering from typhoid, typhus, and other forms of exanthematous fevers, more especially small-pox. The appearance of tyrosin and leucin in such cases not being due, as some (I think) have erroneously supposed, to the exanthematous affection *per se*, but to the disordered condition of the liver which so often accompanies these cases. In almost all exanthematous diseases, the liver is more or less gravely affected. Hence I opine that the appearance of tyrosin and leucin in such cases ought to be attributed to the disordered condition of the liver, and not to the exanthematous disease *per se*.

While inducing artificial jaundice in animals, I have at the same time been equally successful in producing both tyrosin and leucin artificially in their urine, even when there was no evidence whatever of either acute or chronic atrophy of the liver having taken place. Namely, in cases of artificial jaundice the result of suppression, consequent upon congestion of the liver produced by blood-poisoning. Moreover, Zuelzer found both leucin and tyrosin in the livers of patients who died of relapsing fever at St. Petersburg in 1864, Frerichs in those who died of typhoid, while others have found these substances in the urine of typhus patients ; but be it remembered that hepatic derangement is common in all of these diseases.

Long before these observations were published I found in the artificially concentrated urine of a case of jaundice from obstruction, consequent upon impacted gall-stone, a few balls closely resembling leucin in shape and size, but differing from it in being excessively dark in colour. No tyrosin crystals were observed, and unfortunately there was not sufficient of the leucin-like substance present in the urine to admit of its being chemically tested. I thought it my duty to record this fact, however, in my monograph on Jaundice (1863) for the benefit of other observers, as there can be little doubt that we are gradually verging towards some important discovery in a

diagnostic point of view in connection with these substances.

As so very little is known regarding the pathological significance of tyrosin and leucin, I think it may be as well for me to mention that Dr. Charles Anderson, in a paper read before the Medico-Chirurgical Society (1880), stated that he found that tyrosin and leucin appeared in the urine in one case of heart disease, one of asthma, one of bronchitis, one of delirium tremens, and one of hemiplegia, as well as in a number of other affections apparently having no direct connection with the liver. His observations are, however, too vague to admit of any general conclusions being drawn from them.

Before we shall ever be able thoroughly to appreciate the pathological importance and clinical significance of tyrosin and leucin in disease, it will be absolutely necessary for us to learn something more than we as yet know of their origin and physiological modes of action. At the present moment, most physiologists are of opinion that although these substances are not normal products of the liver, they are probably formed by it in a state of disease out of protein substances. Kühne found that trypsin, the proteolytic ferment of the pancreas, acting on albumen, gives origin to both tyrosin and leucin; while no tyrosin and but

very little leucin is to be met with in the absolutely fresh juice of the pancreas. From which fact he concludes that these substances are neither of them normal constituents of the pancreas, but are due to an abnormal species of auto-digestion, from the pancreatic ferment trypsin acting on the albuminoid tissues of the organ. But, be this as it may, it unfortunately does not answer the question as to how tyrosin and leucin are products of the diseased liver. Radziejewski, while stating that leucin and tyrosin are products of pancreatic digestion, and are under ordinary circumstances decomposed in the intestines, asserts that after a dose of calomel they appear in the fæces, owing, as he thinks, to the mercurial sweeping them out of the bowel before they have had time to become completely decomposed.

Before leaving the subject of tyrosin and leucin I may give what may prove a valuable hint to inexperienced observers. Namely, that when they fail to detect either one or other of these substances in the tissues, they should put the organ aside for a couple of days, and they will find by that time the crystals will form and be readily detectible among the granular débris of probably both the liver and kidneys. At least, such has been my experience.

Melanin in the Urine.

In 1858, Dr. Eiselt of Prague called attention to the fact that in cases of melanotic cancer of the liver, melanin appears in the renal secretion.[1] When the urine is passed it is usually quite clear; but after standing it becomes of a dark colour, even as dark as porter, without, however, losing its transparency. This deepening of the colour is no doubt due to the oxidation of the melanotic pigment, as the employment of an oxidising agent, such as nitric or chromic acid, causes the same change to occur instantly.

In addition to the cases related by Dr. Eiselt, I am able to give one of considerable importance, as it not only offers a striking illustration of the correctness of his views, but has the double advantage of being an unbiassed record of facts, in consequence of its having been observed and recorded long before Dr. Eiselt's views were published, and therefore at a time when I had no idea of its significance. The case occurred in 1851, in the wards of the Royal Infirmary of Edinburgh. The history of the case I extract from my note-book. It is briefly as follows:—In the month of May a sailor was admitted into the clinical wards of the

[1] Dr. Eiselt states that he also found melanin in the urine in a case of melanotic cancer of the eyeball.

Royal Infirmary with symptoms of jaundice from enlarged liver. He stated that he had been a great deal abroad in hot climates, and admitted that he had been a hard drinker. On admission his skin was of a dusky yellow colour, and had been so since the month of February. The liver was considerably enlarged, and he complained of sudden attacks of violent pain in the neighbourhood of the umbilicus. These painful attacks were usually most severe during the night. The urine was of a dark colour, and on the addition of nitric acid became nearly black. It contained no albumen. The patient died ten days after admission. On *post-mortem* examination the hepatic duct was found blocked up with a malignant deposit of melanotic cancer, and the liver was of a dark green colour. There was also a considerable amount of malignant deposit in the mesentery. This patient, as frequently happens in such cases, became delirious before death.

Melanin is one of the animal pigments to which I particularly directed my attention while working in Würtz and Verdeil's practical physiological chemical laboratory in Paris in 1852; and I then discovered that true melanin, like hæmatin, urohæmatin, and biliverdin, contains iron in its composition. In fact, melanin, like all the other animal pigments, is a ferruginous colourless albuminoid pigment,

which, like the blue indigo pigment of the plant, only becomes coloured by oxidation; and moreover, like the vegetable pigment, passes through all the intermediate stages of yellow, green, blue, purple, and red, in direct proportion to the amount of its oxidation. See p. 394, vol. iii. of Robin and Verdeil's 'Chimie Anatomique et Physiologique,' where an account of my experiments will be found. As is there stated, I found 1 per cent. of oxide of iron in the melanin experimented upon.

In jaundice arising from melanotic cancer of the liver, the recognition of the presence of melanin in the urine is an important aid to the diagnosis. Care must be taken not to confound the dark olive-green urine occasionally met with in other forms of jaundice with the melanotic urine just described, which only becomes black after standing, or the addition of an acid, and that too, in general, only when the acid is added to the hot boiling urine. Some urine becomes black on being boiled with liquor potassæ, but that has nothing whatever to do with melanin, and must not be mistaken for it, or both patient and doctor may become unnecessarily alarmed. In illustration of the value of this remark I may refer to a case which fell under my notice while I was resident physician in the Royal Infirmary of Edinburgh in 1850. A woman, aged 28, was admitted with a universal and bright jaundice of three weeks'

standing. Her urine was high-coloured, and of a specific gravity of 1022. It contained a small quantity of albumen, and became perfectly black on being boiled with nitric acid. In this case there was no reason to suspect malignant disease of the liver ; the colour of the urine was, therefore, most probably due to the bile-pigment being more than usually oxidised. After a six weeks' stay in the hospital, I dismissed the patient as cured.

The value of the presence of melanin in the urine as a diagnostic sign, in the hands of those who understand how to detect it, it is scarcely possible to overrate. The history of the following case will, I think, convince the reader of the truth of this remark.

On October 2, 1877, a celebrated Q.C. consulted me about the nature of what he called an 'inveterate indigestion.' He told me he had consulted four physicians, among whom he named Drs. Andrew Clarke and Murchison, and that they all agreed that it was a simple case of 'functional derangement.' I listened most carefully to all he said, examined his abdomen to the best of my abilities, but found nothing—not even so much as an iota of a suspicious physical sign. Yet I could not reconcile the 'inveterate indigestion' with the idea of 'mere functional derangement'—perhaps for the simple reason that 'functional derangement,' as a disease, occupies no place in my medical nosology. At any rate I acted

according to my invariable principle, which is—when in doubt, analyse the urine. The urine was brought. I analysed it, and found therein a trace of melanin ! The diagnosis was made—'malignant disease of the stomach.' It is said that one swallow does not make a summer. But such a line of reasoning does not hold good in disease. To my way of thinking the undeniable existence of one single cancer-cell, be it where it may, or the tiniest trace of melanin, is proof positive of the existence of malignant disease, which a million of negatives cannot gainsay. This has been proved to me many times, but seldom or never more conclusively than in this case, unless it be in one other equally striking, if not more extraordinary, where a few cancer-cells in the urine gave a correct clue to the nature of a most obscure case of cancer in a dignitary connected with Westminster Abbey, whom I frequently saw in consultation along with Mr. Holthouse, and who was also seen by Drs. Bence Jones and George Johnson, as well as by Mr. Prescott Hewitt. The Q.C. whose case I am now referring to expected shortly to be raised to the Bench, and he asked me to be plain-spoken to him and hide nothing. Accordingly I was so, and honestly told him that with care he might live a year or two, but without care he would not live six months. He thanked me, and by my advice went back to Dr. Murchison (who happened to be the physician who was

then prescribing for him) with a note from me, telling him of my having found melanin in the urine, and explaining to him how to test for it. Back came the Q.C. in a week, somewhat sarcastically saying, 'How you doctors differ ! not only in matters of opinion, for that would be nothing, but in actual matters of fact. Murchison has three times examined my urine, and he can't find a vestige of melanin in it, though you said that you found it at once. He says that with all your chemical knowledge you have mistaken altered urine pigment for melanin, and that, instead of my having a deadly disease about me, he will cure me in three weeks.'

Too well, alas ! I knew not only the value to be attached to the promised cure, but that the cause of Dr. Murchison's not finding melanin arose from his confounding the reaction of so small a quantity with that of urohæmatin. Therefore I expressed to the patient no opinion regarding Dr. Murchison's remarks, but simply said : 'As he says that he can cure you in three weeks, and I feel sure that I cannot cure you at all, go back to him and let him treat you up till New Year's Day. That is six weeks from now—exactly double the time which he says will suffice for your cure—and if you are not cured by that time come back and put yourself unconditionally under me. In any case, come and see me on New Year's Day, and I sincerely hope to be

able to congratulate you on Murchison's success.' He said he would follow my advice, and so we parted, but I never saw him again until February 15, 1878—three days before his death—when he sent for me, as he said, in order to receive my forgiveness for having broken his promise to me. Our interview was to me a most painful one. His whole conversation being a narrative of blighted hopes, and vain regrets at having put faith in the promised cure, at the same time telling me how he had been kept buoyed up with hope until exactly three weeks before that day, when Murchison informed him that he had a cancerous tumour of the pylorus. I put my hand to the spot indicated, and there, sure enough, was a tumour as big as a cricket-ball. This, then, is another case illustrative of the vast importance of introducing science into the domain of practical medicine. Be it noted, too, that the patient lived only four and a half months from the time that I said with care he might live a year or two, and without care not six months.

In concluding these remarks I have only further to observe that while the absence of melanin from the urine in no case negatives the probability of melanotic cancer, its presence is indubitable proof of its existence. And before a practitioner ventures to give a decided opinion either regarding its presence or absence, he must have assured himself that

he is in a position to recognise the reaction of the substance when it is actually present, or he will most probably repeat the very same error, both as regards diagnosis and treatment, that was committed in the above case.

Urea and Uric Acid.

The presence and quantity of certain other substances met with in the urine of jaundice, although not peculiar to that condition, nevertheless afford us important information, not only as to its cause, but also as to its probable mode of termination. For example, a correct knowledge of the quantity of urea and of uric acid passed in the twenty-four hours is of great value. This can be readily understood if it be true, as Voit believes, that urea is produced in the liver through the disintegration of the hæmoglobin of the effete red blood-corpuscles. But I must candidly admit that not only all my experimental data, but likewise my general knowledge of the origin of urea, go to prove that it is not a special product of the liver, but is the ultimate product of all tissue disintegration; and consequently the quantity eliminated from the body fluctuates in exact proportion as the activity of life fluctuates.

Vogel states that the amount of urea in the urine is diminished in hepatic cancer, being sometimes no more than 246 grains in twenty-four hours. A statement which Julius Jacobs flatly contradicts.

I now say, as I said in my book on Jaundice in 1863, that it is diminished.

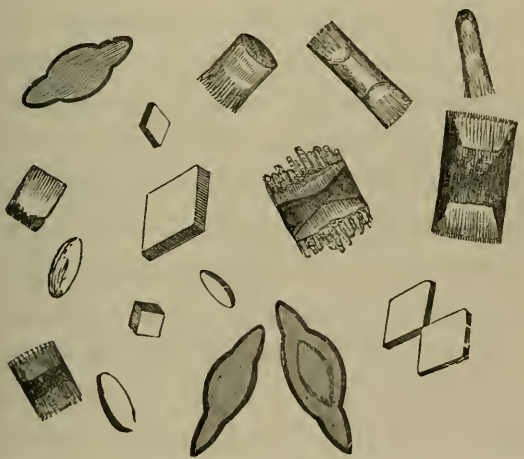
But I must remark that the whole subject requires working out, for even as regards different stages of the same disease it appears that very great differences exist in the quantity of urea excreted by the kidneys.

Dr. Ralfe makes the following remarks in connection with a case of acute yellow atrophy of the liver, reported in the 'Lancet,' vol. i. 1881. After pointing out that some observers have stated that the urea is increased, and others that it is not affected, whilst the majority assert that it is considerably diminished, he says that these statements, though apparently contradictory, are not so in reality, since the differences in the excretion of urea in acute yellow atrophy depend, most probably, on the stage of the disease when the examination of the urine is made. An increase in the quantity of urea excreted would be naturally expected at the commencement of the disease, from the disintegration of the nitrogenous elements of the liver itself. As the disease progresses, the quantity would gradually diminish, till, when the process of tissue degeneration was complete, the urea would be very considerably diminished. Thus in two cases of acute yellow atrophy which were under Dr. Ralfe's care, in one ('British Medical Journal,' vol. ii. 1878)

the urea was but little diminished, whereas in the other it was very considerably diminished. The former ran a rapid, the latter a slow course; the boy having been jaundiced for five weeks, and the acute symptoms lasting more than seven days.

At present I think it may be at least said that in very many cases the amount of urea eliminated

FIG. 18.



Crystals of Uric Acid from Human Urine.

in the twenty-four hours with the urine diminishes in proportion to the destruction of liver-tissue; for this has been observed to occur, not alone in cases of encephaloid, but also in those of suppuration of the liver, where the whole secreting structures have been almost entirely destroyed.

In cases of hepatitis or active congestion, on the other hand, in consequence of the activity of the

hepatic functions being increased, there is an excessive elimination of both the uric acid and urea (Parkes). This fact is readily accounted for by the result of researches made by Meissner,[1] which show that both uric acid and urea to some extent are formed in the glandular tissue of the liver; observations that have been experimentally confirmed by Cyon.[2]

As regards the absolute amount of uric acid said to be found in the urine in cases of hepatic disease, there are nearly as many contradictory observations as regarding the urea, and the reason is very simple. The observations, though made on cases associated with jaundice, have been made on cases which were as different from each other in their pathology as night is from day. Moreover, as I showed in the physiological chapter, from the liver's composition fluctuating in health at different times according to the kind and quantity of the food taken, we cannot expect that it will remain stationary in disease. To show the justness of this remark, I may mention that while I found only three and a half grains in the twenty-four hours' urine of a permanent case of jaundice, Dr. Foot ('Dublin Journal of Medical Science,' 1876, p. 478) found no less than twenty-seven grains! Showing that if the analyses were equally correctly made,

[1] Henle's *Zeitsch.* Bd. xxxi. [2] *Centralblatt*, 1870.

the pathology of the two cases must have been very different. And no doubt their pathologies were totally different, for the following reasons :— In the first place, in Dr. Foot's case, the attack began with great pain in the region of the liver, especially towards the back, which was diagnosed, and I have no doubt correctly, as gall-stones. In the second place, at the time the analysis of the urine was made, the liver was both enlarged and tender. In the third, neither tyrosin nor leucin was present in the urine. In the fourth, the urea amounted to 439½ grains. While lastly there was marked xanthelasma planum associated with it. Above all, the enlargement of the liver forms a marked point of contrast with the atrophied condition the organ presented in my case. So as Dr. Foot's case presented only one feature in common with mine, namely, the permanency of the jaundice (three years' duration), one cannot feel surprised at his patient passing twenty-seven, and mine only three and a half, grains of uric acid in the twenty-four hours. The analysis of my patient's urine is given at pages 773 and 778.

One important clinical fact in connection with the quantity of uric acid eliminated in hepatic diseases has, I think, been sufficiently clearly demonstrated. Namely, that in the majority of cases of fatal permanent jaundice, where there exists no

cancer, the uric acid is diminished. I must here warn the reader against attempting to calculate the amount of uric acid eliminated by a mere naked-eye inspection of the urine, on the ground that crystals of uric acid often form spontaneously when a smaller than normal quantity is actually being passed. This arises from the fact of the urine being in small quantity, and from uric acid being sparingly soluble in water. Indeed, no one can have failed to notice both free crystals of uric acid and a copious

Fig. 19.

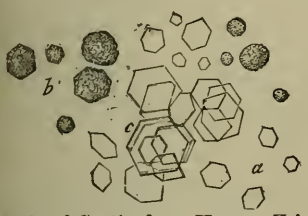
Crystals of Oxalate of Lime.

deposit of urates in many cases of hepatic disease; for even when they are being passed in normal quantity, if the urine is slightly below the average amount, their presence becomes manifest as soon as it gets cold, from the fact of both of these substances being insoluble in cold, though readily soluble in hot, urine. Consequently, whenever the patient is passing less than thirty ounces of water per diem, no importance is to be attached to the

appearance of a moderate amount of uric acid or urates in the cold urine.

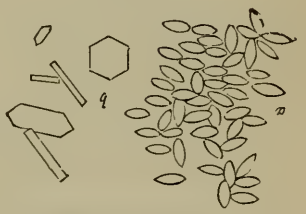
When all the functions of the liver are in perfect order, its oxidising and transforming processes go on so perfectly, that highly soluble urea is their ultimate result. Whereas, when these processes are incomplete, the transformation change is cut short at the formation of uric acid, or some one or other of the manifold intervening stages between the higher and lower forms of effete products—to wit, hypoxanthin, leucin, tyrosin, cystin, oxalic acid, &c. &c.,

FIG. 20.



Crystals of Cystin from Human Urine.

FIG. 21.



Hypoxanthin from Human Urine.

together with a concomitant excessive elimination of carbonates. In illustration of this remark I may mention that taurin, that remarkable product of the liver which is one of the normal substances met with in the human body containing sulphur, is transformed into the abnormal substance named cystin when the oxidising and transforming processes of the liver are deranged, and the cystin—which also contains sulphur—is eliminated as a foreign material by the kidneys. Oxalic acid, in like manner, under a precisely similar set of circumstances, appears in the

urine in the form of insoluble oxalate of lime in the course of a great variety of hepatic affections.

Neither the physiology nor the chemistry of the various hepatic oxidising and transforming processes is as yet understood; which is not surprising, seeing how many and how complicated are the substances and their products with which the liver has to deal. A glance at the subjoined table will suffice to show this :—

Glycocholic acid, $C_{26}H_{43}NO_6$.
Taurocholic acid, $C_{26}H_{45}NSO_7$.
Cholesterin, $C_{26}H_{44}O$, H_2O.
Urea, $CH_4N_2O = CO\left<\begin{matrix}NH_2\\NH_2\end{matrix}\right.$
Uric acid, $C_5H_4N_4O_3$.
Hypoxanthin, or
Sarcin, $C_5H_4N_4O$.
Leucin, $C_6H_{13}NO_2$, or
Amido-isocaproic acid, $C_5H_{11}(NH_2)COOH$.
Tyrosin, $C_9H_{11}NO_3$, or
Oxyphenyl-amido-propionic acid, $C_6H_4\left<\begin{matrix}OH\\C_2H_3(NH_2)COOH\end{matrix}\right.$.
Taurin, $C_2H_7NSO_3$, or
Amido-ethene-sulphonic acid, $C_2H_4\left<\begin{matrix}NH_2\\SO_3H\end{matrix}\right.$.
Cystin, $C_3H_7NSO_2$, or $C_2H_3(SH)(NH_2)COOH$.
Hippuric acid, $C_9H_9NO_3$, or
Benzamid acetic acid, $CH_2\left<\begin{matrix}NHC_7H_5O\\COOH\end{matrix}\right.$.
Oxalic acid, $C_2H_2O_4$, or $C_2O_2\left<\begin{matrix}OH\\OH,2H_2O\end{matrix}\right.$.

It will be observed that three of the above substances are sulphur compounds. To the liver, therefore, we must look as the source of the sulphur-containing urinary calculi, whose origin was so long a puzzle to urologists.

The experiments of Salomon ('Zeitschrift für Phys. Chem.' 1877–8, p. 90) and Chittenden ('Jour. of Phys.' 1879–80, p. 28) show that, besides uric acid and urea, hypoxanthin, another substance which forms urinary calculi, is a decomposition product of blood-fibrin. Blood-fibrin, after being in simple contact with boiling water for from twelve to twenty-four hours, yields appreciable quantities of hypoxanthin, and when blood-fibrin is acted upon by pancreatic juice, it is not only changed into hypoxanthin, but into leucin and tyrosin as well.

The value of the preceding remarks regarding the amount of urea, uric acid, &c., daily eliminated, will be best appreciated by giving a short account of a case of obscure disease where a correct diagnosis and prognosis could not have been arrived at without the application of the chemical knowledge referred to. I shall, therefore, relate briefly the history of a typical case, the diagnosis of which was solely arrived at by chemical means, for the very good reason that none other were of any avail in unravelling its puzzling symptoms.

The case was that of a gentleman, aged 50, who had been a remarkably healthy man until within eighteen months of his death, when for the first time he noticed that his skin was gradually assuming a more and more jaundiced tint, without, as he thought, any assignable cause. The stools were pipeclay-

coloured, and the urine loaded with bile-pigment. Soon after he noticed the discoloration of the skin he began to lose flesh. The liver became enlarged, and somewhat tender to the touch ; the gall-bladder being at the same time so distended that it could be seen, as well as felt, projecting from under the false ribs. As the case resisted the usual remedies, the patient was recommended to try change of air. During his absence from town, he suddenly passed a large quantity of yellow matter by stool (supposed by him to be bile), and immediately afterwards the swelling in the abdomen disappeared. On the patient's return to town, the gall-bladder could no longer be felt, and it was naturally supposed that it had emptied itself on the occasion referred to. As, notwithstanding this, the jaundice continued, and the health and strength gradually declined, Dr. Prance, of Hampstead, under whose care the patient was, sought the assistance of a physician of distinguished reputation in these affections. At this period, however, the entire absence of physical signs beyond the clay-coloured stools, and those directly referable to the jaundice, rendered it impossible for Dr. Prance, or the consultant who then saw him, to form any very decided opinion regarding the nature of the case.

At the time now alluded to, the liver, from having been at first enlarged, had resumed its normal size, and the only points ascertainable on physical

examination were a slight tenderness of the organ on firm pressure, and a somewhat doubtful fulness in the pancreatic region. (See p. 114.)

These signs, associated as they were with gradually increasing emaciation and debility, led to the suspicion of malignant disease, either in the course of the bile-ducts, or at the head of the pancreas. About this time it was discovered that the patient occasionally passed a considerable amount of a fatty-looking matter by stool—not mixed with the motions, but separate, though lying upon them. After the passage of this matter, there in general appeared to be a slight improvement in the patient's condition. The substance in question, on cooling, solidified into a firm pale-brown matter, resembling Old Brown Windsor soap, and not at all unlike a solid mixture of chyme with some of the biliary products. This led to the idea that it might partly be composed of the fats of the bile and chyme. So on one occasion a portion of it was forwarded to me for analysis, and on subjecting it to chemical examination I at once discovered that it was modified fish-oil, the olein of which had entirely disappeared. In fact, on further analysis and enquiry regarding what the patient was taking, it turned out to be nothing but the sparingly soluble fatty acids of cod-liver oil, which had been modified during the process of digestion in the stomach, and from which all the olein

liquid principles had been absorbed. This was considered an important discovery, as it not only negatived the idea of the bile still reaching the intestines, but also proved that the *pancreas*, as well as the *liver*, was affected. Having thus learned that the pancreatic juice, as well as the bile, failed to reach the intestines, an effort was at once made to counteract the pernicious effect on the system caused by the absence of the former secretion, by giving $1\frac{1}{2}$ grains of pure solid pancreatin in the form of pill three times a day. During the period that the patient was taking this medicine, the quantity of fat passed by stool was supposed to diminish. No decided improvement in the patient's condition, however, took place, and on November 2 the gentleman was brought to me by his medical attendant, in order that I might personally examine him, which I had not as yet done. At this time the patient was much in the state already described, and I noted his condition to be as follows :—Skin of a black jaundiced tint (dark green). Eyes deeply stained. Lips anæmic. Considerable emaciation and debility. Extreme languor. Appetite good. Tongue and pulse not remarkable. Slight pain on pressure over the gall-bladder. Indistinct fulness in pancreatic region, and to the left of middle line. The hepatic dulness was perfectly natural; there was no tenderness to speak of, and no distinct history of gall-stones, while the only evidence of

tumour was the doubtful sense of fulness on palpation in the pancreatic region. The digestive and other functions of the body, except those already mentioned, seemed unimpaired, and yet the patient's strength daily declined. As physical as well as symptomatical diagnosis proved inadequate to unravel the mystery of this obscure case, and as chemistry had already, in as far as it had been tried, been of advantage, it was resolved to subject the excretions to a rigid chemical examination. The patient was accordingly desired to collect all the urine he passed during twenty-four hours, and while I analysed it, Dr. Prance daily examined the stools with the naked eye, in order to ascertain their probable composition—especially as regarded the amount of fatty and albuminous matters contained in them.

The urine yielded on analysis the following result :—

TWENTY-FOUR HOURS' URINE.

Quantity (55 oz.)	1705 cc.
Reaction	Acid
Specific gravity	1018
Colour	Greenish-yellow
Urea	27·28 grammes
Uric acid (crystals large, and of a dark-green colour)	0·511 „
Bile acids	Abundant
Bile pigment [1]	Abundant
Albumen	None
Sugar	None

[1] Nitric acid at first turned the urine green, but on the application of heat it became red, and, after prolonged boiling, of a pale straw colour. Hydrochloric acid changed the colour of the urine immediately to a deep olive-green tint.

The facts elicited were interpreted as follows :—

1st,—The quantity of urea, which might be said to be normal, was considered a favourable sign.

2nd,—The quantity of uric acid being below the average was likewise regarded as favourable, tending as it did to negative the idea of cancerous disease of the liver. Uric acid being said to be in cases of cancer of the liver usually increased, a fact about the value of which I have, however, still some slight doubt.

3rdly, and lastly—The presence of the biliary acids, as well as the bile-pigment, in the urine, showed that bile, though still being secreted, was not being excreted, but retained and re-absorbed. This fact at once led to the diagnosis that the case was one of jaundice from obstruction.

Here, then, was another important step gained. The next point was, if possible, to ascertain the cause of the obstruction. Taking into account the absence of any history of gall-stones, together with the fact of the sudden disappearance of the enlarged gall-bladder, my first idea was that it might be a case of hydatids blocking up the common bile-duct, and that on the occasion when the sudden discharge of bile took place the cyst had ruptured, and discharged itself through the intestines, and at the same time allowed the gall-bladder to empty itself. On talking the case over with Dr. Prance, however, that idea

was abandoned, and we were forced to content ourselves with the simple conclusion that the case was one of jaundice from obstruction of the common bile-duct, complicated with occlusion of the pancreatic duct, which fact had been previously ascertained by the discovery of the fatty acids in the fæces, and was subsequently verified by the result of the autopsy. About this time the patient took three grains of benzoic acid, in the form of pill thrice a day, and, it was thought, with the advantage of slightly diminishing the jaundiced state of the skin. But no permanent benefit was obtained, and after a time this remedial agent had to be discontinued, in consequence of its having induced slight dyspepsia. In the letter I received informing me of this fact, it was also noted that there was much less both of the oily matter and albumen in the stools. There was, at the same time, a considerable deposit of urates in the urine. The specific gravity continued to be about 1018. The quantity in twenty-four hours about forty ounces. On November 29, the patient was again brought to me, and we made a careful examination of the size, shape, and exact position of the hepatic organ. Its perpendicular measurements were found to be 5 inches in the right axillary line, 4 inches in a line drawn perpendicularly to the nipple, and $2\frac{3}{4}$ inches midway between nipple and sternum. Beyond the centre of the sternum the liver did not reach. Hence it was

concluded that the liver was somewhat atrophied; for the patient was fully six feet high.

On this occasion it was observed that the patient's memory was not so good as formerly, and that there was also a certain amount of mental as well as bodily languor. His hearing was likewise sluggish, words having occasionally to be repeated before they made an impression on the cerebral organ. This, no doubt, arose from the poisonous effects of the bile circulating in his blood. (See p. 739.)

It may here be mentioned that six grains of pure glycocholate of soda killed a small dog, into whose femoral vein I injected it, in the course of two hours.

In experimenting on animals, I have made the curious observation, that although bile has the property of retarding or arresting putrefaction, both in the intestinal canal and out of the body, yet, when injected into the subcutaneous cellular tissue of a healthy animal, it causes the surrounding tissues to decompose and become fœtid, and an artificial disease to be set up, whose most peculiar feature is the engendering of a rapid putrefaction of the whole body after death. (See p. 741.)

In cases of jaundice from suppression we do not often meet with those extreme symptoms of cerebral disturbance which are so common in cases of jaundice from obstruction. I believe the reason of this difference in the two forms of jaundice arises from

the circumstance that the really most virulently poisonous parts of the bile are the biliary acids, and that they, like urea, are powerful narcotic poisons. The results of my experiments on artificial jaundice led me to this conclusion.

The views I published in 1863 regarding the poisonous effects of the bile acids upon the nervous system have since then been fully borne out by the results of the experiments of Feltz and Ritter, published in Robin's 'Journal de l'Anatomie et Physiologie' for 1875. They found that when the bile acids obtained from ox bile were injected into the veins of dogs they acted as violent cerebral poisons. The blood-corpuscles were destroyed, and their contents eliminated with the urine, as well as the bile acids themselves. Hæmorrhages from the mucous membranes also occurred. The injection of cholesterin produced no such effects, while that of bile-pigment produced only a lowering of the temperature, obstinate constipation, and an increased flow of urine, but no cerebral symptoms. The injection of fresh bile itself into the veins was followed by its rapid elimination by the kidneys, salivary glands, and intestines. After large doses, vomiting, bilious diarrhœa, and bloody urine supervened, while in still larger doses it produced tetanic convulsions, coma, and death. A series of effects exactly the same as those arising in cases of jaundice of varying severity. (See p. 742.)

As neither the symptoms nor the physical signs threw any light whatever on this highly interesting case, it was determined once more to interrogate nature by again bringing chemistry and the microscope to bear upon it, with the view, if possible, of still further extending the information these methods of investigation had already yielded. Accordingly, a specimen of the urine was again obtained for analysis, and it yielded the following results :—

TWENTY-FOUR HOURS' URINE.

Quantity (43 oz.)	1333 cc.
Specific gravity	1016
Reaction	Acid
Urea	23·994 grammes
Uric acid	0·266 „
Bile pigment	Abundant
Bile acids	Only in small quantity
Sugar	A little
Solids (total)	41·989
Organic matter	31·992
Inorganic „	9·997

A marked change is here seen to have occurred in the constitution of the renal secretion. For, first, the quantity of urea has notably diminished, from 27·28 to 23·99 grammes, in other words from 423·84 to 370 grains. The amount of uric acid has also fallen from 0·511 to 0·266 gramme, or, in other words, from 8 to 4 grains. At the same time the biliary acids have considerably decreased, while the quantity of bile-pigment remains almost the same. These changes are also seen to be accompanied by another,

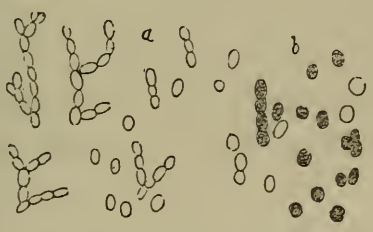
which I at once regarded as a most unfavourable sign—namely, the appearance of sugar in the urine.

Sugar in the Urine.

Although the quantity of sugar was as yet small, and it was associated with a diminution in the bile acids, it nevertheless made me look forward with gloomy forebodings, for, as far as my experience goes, the appearance of saccharine matter in the urine, in the course of a chronic and exhausting disease, is generally the forerunner of a fatal termination.

Although present in only small quantity, it was nevertheless sufficiently abundant to spontaneously

FIG. 22.



Torulæ Cerevisiæ from Saccharine Urine.

ferment, and after standing forty-eight hours, abundance of torula spores were found in it when a drop of the deposit which had formed in the urine glass was examined with the microscope.

This case, I am sorry to say, proved no exception to the rule that the appearance of even a trifling

glycosuria of a permanent character during the course of severe chronic disease is a sign of bad omen.

In connection with this subject I may mention that Dr. Legg ('St. Bartholomew's Hospital Reports,' 1873) says that he found that after he had imitated the effects of jaundice from obstruction by placing a ligature on the bile-duct, glucogen disappeared from the healthy liver of the animal operated upon ; and to this he adds the still more unaccountable observation, that irritation of the fourth cerebral ventricle (which under ordinary circumstances would have caused glycosuria) failed to produce any such effect. Theorising on these observations, he says he thinks that in cases of jaundice from obstruction, sugar *ought not to be found* in the urine. Such a theory is in direct opposition to my own experiences in the human subject, as well as to those of Golowin in dogs ('Arch. Path. Anat.' 1871, p. 428).

To return to our patient. There was, indeed, but one consolatory fact in the last analysis, and that was the diminution of the uric acid, which, as I before remarked, tended, it was thought, to negative the idea of malignant disease of the liver ; and this was a great source of satisfaction to the patient and his friends, who had been alarmed by the opinion expressed by the first consultant.

Eight days later (November 12) a qualitative and quantitative analysis of the urine was again made, with the following result :—

TWENTY-FOUR HOURS' URINE.

Quantity (33 oz.)	1023 cc.
Reaction	Acid
Specific gravity	1017
Urea	15·345 grammes
Uric acid	?
Bile acids	None
Bile pigment	Abundant
Sugar	Increased
Leucin and tyrosin	In distinct, though in small quantity only
Solids (total)	23·426
Organic matter	17·698
Inorganic „	5·728

Here is now to be observed the rapid downward progress of the case. Stomachal digestion, as indicated by the amount of urea, is much impaired. The general health, as indicated by the sugar, is sadly affected, and, to crown all, tyrosin and leucin, the decided indicators of atrophy of the liver, have made their appearance. On precipitating the urine with the acetate of lead, filtering, and freeing the clear liquid from the excess of that reagent by means of sulphuretted hydrogen, and again filtering, in the liquid, after evaporation, was found a deposit of small crystals of tyrosin, while floating in and on its surface were round balls of leucin. This discovery of tyrosin and leucin in the urine was a most important one, from the fact that until it was made on this occasion no one knew that these substances were to be met with in any form of liver atrophy except one,

namely, acute or yellow atrophy. So I naturally enough saw that my finding both tyrosin and leucin in this case had a wide pathological significance, as it suggested not only that Frerichs's ideas of their pathology would possibly require considerable modification, but that we should most probably meet with them under still more decidedly different pathological conditions. An idea which has since been verified.

So unfavourable was the result of this analysis considered, that Dr. Prance felt himself bound to fulfil a promise he had made to the family some time previously to warn them of approaching danger so soon as we had no longer hope of the patient's recovery.

Some time afterwards, in the beginning of December, we again saw the patient together, and made a physical examination of the liver, the result of which only confirmed our suspicions. The organ was already decidedly smaller. The epigastric tenderness increased. The jaundiced tint deeper. Petechial spots had now appeared on the trunk and arms. The lower extremities were œdematous, and the abdomen two-thirds filled with fluid.

On December 31 I received a sample of urine, and a note saying that the patient had slightly rallied. But on examining the urine, it was found to have a neutral reaction—it had previously been

acid—to be of a specific gravity of 1019, and, on standing, to deposit a copious sediment of lithates, coloured intensely yellow, not pink, with the bile-pigment. The sugar had increased.

A few days later, and just before his death, the patient had the benefit of another physician's opinion, Dr. Bence Jones being asked to meet us in consultation. Although his opinion entirely differed from the foregoing, it was, nevertheless, equally unfavourable, for he considered the case to be one of malignant disease of the liver, as it had been originally diagnosed.

The gentleman, having noticed that his symptoms excited considerable interest and some difference of opinion among his medical attendants, directed that his body should be examined after death; and as this wish was seconded by his wife, a lady of superior mind and accomplishments, a *post-mortem* examination was accordingly made, with the following results.

Morbid Anatomical Conditions in a case of Slow Obstructive Jaundice.

First,—The orifice of the common bile-duct was completely obliterated (Plate I., *f*, p.113), and the duct itself immensely distended with dark thick tarry bile, which on microscopic examination was found loaded with beautiful crystals of cholesterin, as represented

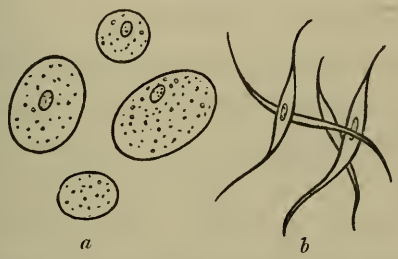
at fig. 14, p. 551. I have occasionally, though rarely, found crystals of pure cholesterin in the urine of hepatic cases, and although this at first surprised me, on account of cholesterin being insoluble in water, it does so no longer, seeing that crystals of it are met with in such a variety of circumstances, and in so many different situations. To wit, hepatic hydatid fluids, ovarian fluids, cystic fluids, pleuritic fluids, abscesses, tumours of the brain, as well as in atheromatous, fibrinous, and epithelial growths in various parts of the body.

Secondly,—Both hepatic and cystic ducts were proportionally dilated, and equally full of the same tarry thick bile.

Thirdly,—The liver was small in size, excessively dense, even hard to the touch, and very heavy. Externally, it had a dark green olive hue, and on section presented a most curious appearance. The cut surface of the section was of an almost uniform yellowish-green colour, and studded over with excavations (Plate I., *b*), from which thick tarry bile slowly streamed in all directions. The apparent excavations were nothing more or less than immensely distended ducts. On looking into the ducts it was observed that they presented all the appearance of possessing valves, and I satisfied myself that they actually did. On microscopical examination, the hepatic cells were found smaller than normal, as

if partially atrophied. The nuclei were unusually well marked, in consequence of fat-granules being almost entirely absent. (Fig. 23, *a*.) In the field of the microscope were a number of caudate or spindle-

FIG. 23.



a, Altered Hepatic Cells. *b*, Spindle-shaped Connective-tissue Cells.

shaped cells (fig. 23, *b*) from the epithelial lining of the ducts. In the hepatic tissue were found some beautiful stellate crystals, as well as a number of separate needles of tyrosin. A few small crystals of cystin were also seen.

While on the anatomical conditions in cases of permanent obstruction to the outlet of the common bile-duct, I may as well here refer in general terms to what is frequently observed to be the condition of the liver in cases of death arising from long-impacted gall-stones, as they not only differ from the foregoing, but are in many respects even more characteristic of the effects of the backward pressure of the pent-up bile, and their perusal will perhaps give the reader a clearer idea of its true pathology. I may remind

him that the condition met with entirely depends on the duration of the obstruction ; for while it is enlarged, engorged with blood, and reddish yellow in the first, it is in the last stage small, shrunken, even atrophied sometimes to little more than one-third its natural dimensions, and of a deep black-green colour. The hepatic ducts again, while only visible in the first, are large dilated tubes in the last stage, sometimes with bulging extremities; resembling cysts, as big as walnuts, full of thickened tarry-looking bile. Again, while the secreting cells seem to be increased both in size and in number in the first, they appear to be decreased in both respects in the last stage, and thus give rise to the appearance of the connective tissue being absolutely as well as relatively increased in quantity. Relatively increased I admit it must be ; but absolutely increased I am inclined from the results of my own observation to deny. Perhaps it may be so in some cases, but assuredly it is not so in all. Except at least in those where there is a marked hardened as well as a simply atrophied condition of the organ.

Fourthly,—The patient's gall-bladder was enlarged to the size of a swan's egg, and choke-full of thick tarry viscid bile.

Fifthly,—In the abdomen was a considerable quantity of dark yellow straw-coloured serum, which on the addition of strong sulphuric acid became of

a fine emerald-green colour, in consequence of the presence of bile-pigment. Traces of sugar were also present in this effused liquid. The serum had only collected in the latter weeks of the patient's life, and after a decided shrinking of the liver was observed to have begun. The gall-bladder, duodenum, abdominal parietes, and in fact all the abdominal viscera, were more or less intensely stained—some almost blackened—by the osmosed bile.

Sixthly,—The pancreatic duct, as had been suspected from the result of the chemical examination of the fæces, was found to be as completely occluded at its outlet as the common bile-duct. Moreover, it was found so distended by the pent-up pancreatic secretion, as when empty readily to admit the little finger.

Seventhly,—The kidneys were enlarged, pale, and fatty, as is represented in the chromolithograph, Plate II. p. 728. While all over the surface of the section, as well as immediately under the capsules, which were very loosely attached, were small abscesses. The surface was also studded with numerous dark bile-pigment points, and probably the abscesses were the result of the blocking up of the capillary vessels by the pigment deposit, as previously alluded to at page 733.

Eighthly,—The head of the pancreas was considerably enlarged, and on cutting into it, a quantity

of pus oozed out from an abscess in its interior. The abscess was found to communicate with a large cicatrised ulcer in the duodenum, which in cicatrising had occluded both the orifice of the bile and pancreatic duct, and thus produced all the described signs and symptoms. (Plate I., *f.*) On microscopical examination, the tumour of the pancreas was found to consist of an hypertrophy of the normal gland tissue, being, in fact, a chronic inflammatory tumour of the gland substance.

In no portion of the body was so much as a trace of cancer detected, nor any enlargement of the mesenteric or other glands, even to justify the remotest suspicion of the case being one of malignant disease. So the opinion arrived at regarding the pathology of this case is, that the disease originated in an inflammatory affection of the pancreas ending in an abscess which opened into the intestines by ulceration, and during the progress of the cicatrisation of the ulcer, the openings of the bile and pancreatic ducts became blocked up. The interruption to the excretion of the bile giving rise to the jaundice, and at the same time inducing engorgement and enlargement of the liver. The inflammatory affection of the pancreas with its abscess, by pushing the enlarged liver forwards, admitted of the distended gall-bladder being readily seen, and felt through the abdominal parietes. At length the abscess burst,

and suddenly emptied itself into the duodenum ; the yellow fluid discharged from the intestines being not pure bile, as the patient had supposed, but pus mixed with bile. No sooner had the abscess emptied itself, and the liver returned to its natural position, than the emptied gall-bladder suddenly ceased to be seen or felt. The ulcer in the duodenum appeared to be the spot at which the matter was discharged—in fact the mouth of the abscess. Once the occlusion of the orifice of the common bile-duct was complete, the slow and gradual atrophy of the liver would arise from the continued pressure of the distended bile-ducts interrupting the hepatic circulation. Lastly, there being no bile or pancreatic juice admitted into the intestines, the greater part of the food taken passed out of the body unabsorbed, and the patient, though possessing an excellent appetite, and taking plenty of food, actually died of slow starvation.

The presence of bile acids in the urine in the earlier, and their disappearance from it in the later stages of the disease, are easily accounted for, on the natural hypothesis that as long as the hepatic cells were able to perform their functions they manufactured the acids ; but as soon as their functions, in consequence of their compression, became abolished, the bile acids ceased to be formed, and consequently none were found in the urine. While it was quite another thing with the bile-pigment ; for from its being formed in the

circulation, and only excreted by the liver, its manufacture went on unimpeded, and hence there was always abundance of it in the urine up till the time of the patient's death.

Having before explained the mechanism of the two forms of jaundice—that arising from suppression, and that induced by obstruction—it only remains for me here to remind my readers that there is frequently, as was no doubt in this case, a distinct progressive combination of these two conditions. For jaundice from obstruction cannot in any case long exist without becoming complicated with jaundice from suppression. The continued pressure exerted on the hepatic parenchyma by the over-distended bile-tubes sooner or later impedes the circulation in the organ, to an extent sufficient to induce an impairment, if not an almost total arrest, of the biliary secretion. Hence, as has just been said, is explained why, in the last stage of jaundice from obstruction, the biliary acids gradually diminish, and at last finally disappear from the urine. Fortunately, nevertheless, we have it still in our power to distinguish, in the lifetime of the patient, between the two forms of disease. Thus, whereas, in jaundice arising from simple suppression, there is only an absence of the bile acids, in jaundice from obstruction, complicated with suppression, the absence of the bile acids is usually associated with the presence of tyrosin and

leucin. For before complete suppression occurs as a result of obstruction, the hepatic tissue has already had its nutrition so impaired as to admit of the abnormal formation of these foreign substances by the liver itself. Lastly, the history of the case will of itself always be an additional important guide.

My object in giving such prominence to this interesting case is to show clearly how valuable an adjunct physiological chemistry is to the other methods of diagnosis in obscure diseases of the abdominal organs, and to encourage others to follow in the same path. It must be remembered that the foregoing was no mere dead-house diagnosis, but that every fact here stated was discovered and recorded before the patient's death, so that the evidence it furnishes of the value of physiological chemistry in the diagnosis of obscure hepatic disease is undeniable.

Analysis of the Bile taken from the Gall-Bladder.

The bile taken from the gall-bladder was found on analysis to contain in one thousand parts :—

Water			694·45
Solids	Organic	288·99	305·55
	Inorganic	16·56	
			1000·00

Thereby showing a vast difference between it and normal bile—so great, indeed, that I obtained a speci-

men of what was considered to be normal bile from the gall-bladder of a woman aged 61 (the person nearest in age to the patient from whose body I could at the time obtain a specimen). It had a specific gravity of 1020, and contained in one thousand parts :—

Water			933·27
Solids	Organic	56·73	66·73
	Inorganic	10·00	
			1000·00

The difference in composition of the solids of these two biles is very striking. The one contains more than four times as much solid matter as the other; and when the relative amounts of organic and inorganic substances are compared, the curious fact is observed, that the difference in the amount of solids in the two cases is almost entirely due to the change in quantity of organic matter. The inorganic salts have not even so much as doubled themselves in the abnormal bile, while the organic have increased fivefold. Soda is the chief inorganic substance found in bile, and occurs in it in the form of glycocholate and taurocholate of soda, both of which substances are reabsorbed from the distended ducts and gall-bladder into the circulation, from whence they are eliminated with the urine. This fact is no doubt the cause of the inorganic salts being in such small quantity in the abnormal bile of jaundice from obstruction, as is ex-

ceedingly well shown in this case, a more typical than which in every single particular it has never been my lot to meet with.

Hepatic Albuminuria.

Before quitting the subject of the chemistry of the excretions, it may be as well for me to say a few words regarding a form of albuminuria which is a frequent concomitant of hepatic disease, and to call special attention to a simple means of differentiating renal from hepatic albuminuria, which will no doubt be found by the reader useful at the bedside. I know it has on more than one occasion enabled me to make a correct diagnosis where other physicians of justly acknowledged diagnostic skill have fallen into error. Probably it may surprise even some accomplished urologists to be informed that the index alluded to is the very simple one of 'urinary specific gravity.' Strange to say, by paying special attention to that single factor alone, the practitioner may not only be enabled to arrive at a correct differential diagnosis, but, what is of even more importance in a professional point of view, probably be prevented from committing the unfortunate blunder of confounding the effect with the cause of the disease, and treating a healthy kidney instead of a diseased liver.

As nothing is so impressive as personal narrative, I shall briefly relate a case which fully illustrates all

I have to say on the matter, even to its minutest details.

A gentleman from Teignmouth, under the care of the late Dr. Murchison in 1874 for albuminuria, instead of improving rapidly got worse, so Dr. Murchison called and asked me to see the patient along with him. It being, he thought, a very bad case of renal disease. The patient's history was briefly as follows :— He was a gentleman aged about 73, who had resided for thirty years or more in Australia, and always enjoyed good health until within the previous eighteen months. When, while living at Teignmouth, his health began to give way. The first thing he noticed wrong being that his feet swelled and his strength declined. After talking over the symptoms presented by the case, it was arranged that we should meet at the patient's house the same afternoon. On arriving there I found the patient very ill in bed, but complaining more of prostration, with loss of appetite and sleep, than anything else. Before making a physical examination, Dr. Murchison called my attention to the urine, which he tested with heat and nitric acid, and showed me that it was loaded with albumen. In the urine standing in a glass upon the table was a urinometer, and glancing at it I observed that it indicated a specific gravity of 1022, and moreover that, though clear, the urine had a dark amber colour. This made me put the question, ' Is this an average specimen of

the twenty-four hours' urine, or is it only the result of one micturition?' On learning that it was a sample taken from a mixture of the whole twenty-four hours' urine, I at once said, 'This is not a case of kidney disease! If, as you say, there is nothing wrong with the man's heart, it is probably his liver that is wrong.' Dr. Murchison, looking at me with an air of surprise, said, 'What makes you think that?' I replied, 'The specific gravity,' and, seeing that he did not grasp the import of the remark, I added, 'Let us examine the patient's liver.' We at once proceeded to the bed-room and did so, and soon discovered that the liver was slightly enlarged, $5\frac{1}{2}$ inches in the perpendicular nipple line, and tender on firm pressure, especially in the region of the gall-bladder. On being carefully questioned, the patient told us that for a long time past he had felt a dull undefined feeling of uneasiness with a sense of fulness in the right hypochondriac region. Adding that, from always having been accustomed to lie on that side, he had, for the sake of comfort, to turn shortly after getting into bed, and sleep on the left side. There was no jaundice, but the condition of the urine, coupled with the history of the case, led me to the diagnosis of scirrhus of the liver. I said scirrhus, for there was no distinct cancerous cachexia, or I should have said encephaloid. As soon as Dr. Murchison heard my theory of the case, together with my explanation of

the cause of the albuminuria, he at once consented to alter his treatment from that of kidney to that of liver disease, and requested me to prescribe for the patient in my own fashion. I accordingly did so, and gave him a cholagogue cathartic to unload the liver. The effect of which was marvellous. In three days the patient was able to get out of bed, and although to expect a cure was of course out of the question, the functional derangement of the kidneys so greatly improved, that the poor patient, as I was afterwards informed, for I did not see him again, began to think he was getting well. The liver disease, however, steadily increased, and within two weeks before his death (which occurred about ten weeks after I saw him) Dr. Murchison discovered a hard mass in the neighbourhood of the longitudinal fissure. Which, as he said, confirmed his suspicion that the view I had taken of the case two months previously was the correct one.

Now, although the disease of the liver was too far advanced, at the time of our consultation, to admit of much amelioration by the change of treatment from the healthy kidney to the organ that was actually diseased, it nevertheless had the advantage of quickly relieving all the most distressing symptoms, and at the same time, I have little doubt, enabling Dr. Murchison to keep the patient alive for as many weeks as he would probably have done for days,

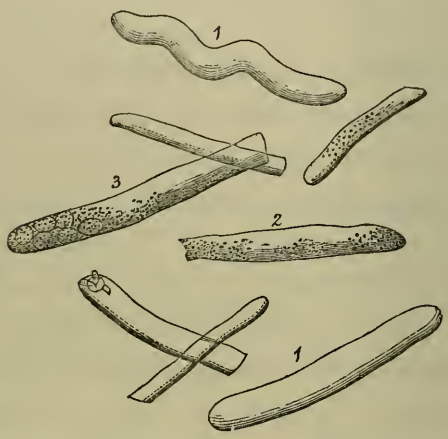
had there not been a prompt change in the treatment.

It may be as well for the benefit of others for me to mention that one, and that too perhaps the chief, reason why Dr. Murchison was so completely misled by the condition of the urine in this case was, as he himself said, on account of his finding granular renal tube casts in the urine, and being unaware that tube casts are quite common even in genuine cases of hepatic albuminuria. Indeed I may mention that granular renal tube casts are not only present (as I have already shown at p. 380) in hepatic hæmaturia, but even in many cases of simply hepatic albuminuria, where there is scarcely more than a trace of albumen to be detected by heat and nitric acid. I have met with both hyaline and granular tube casts (fig. 24). Nothnagel ('Deutsches Archiv für Klin. Med.' Oct. 1873) thinks that renal tube casts always exist in the urine of jaundice when the bile acids are eliminated by the kidneys.

Though the majority of the tube casts are of a granular and sometimes of a hyaline character, they often contain small yellow glistening granules in their interior, and are accompanied by true epithelial renal casts, which are also occasionally stained yellow by the bile-pigment. This is no more than ought to be expected, seeing that the renal cells in the tubuli uriniferi have the elimination of the bile-

pigment thrust upon them in every case of jaundice. No matter whether it be one arising from suppression or from obstruction. But tube casts are no doubt

Fig. 24.



1, Hyaline ; 2 and 3, Granular Renal Tube Casts.

most common in the obstructive forms of jaundice ; that is to say in those cases where bile acids appear in the urine, as Nothnagel observed, but probably not for the precise reasons he gives.

Dr. George Johnson, in his ' Lectures on Bright's Disease,' gives it also as his opinion that desquamated epithelium tube casts appear in the urine in cases of obstructive jaundice from the excess of biliary matter eliminated by the kidneys being so irritative to them as to engender a mild form of nephritis. An idea which I think is a highly probable one. For, as is shown by the lithographed kidney in Plate II. at page 728, the

continual elimination of bile products along with the urine gives rise not only to an inflamed, but even sometimes to a suppurative condition of the organ.

Having thus shown how all-important is the factor of urinary specific gravity in making a differential diagnosis between cases of renal and hepatic albuminuria with and without jaundice, I shall close my remarks on the chemistry of the excretions, and proceed to the second department of diseases of the liver. Namely, the consideration of those not necessarily associated with jaundice. But before doing so, as I think that I have not only redeemed the promise I made, in the Introduction, of adducing evidence to show how valuable an adjunct physiological chemistry is in the diagnosis and treatment of obscure diseases of the liver, but also given a clear, I hope, though succinct account of all the hepatic affections with which jaundice is of necessity associated, it may be well for me now to place before the reader my views of the pathology of jaundice in a tabular form. With but very trifling alterations the table I am about to give is the identical one I published in my work on Jaundice twenty years ago; and as Dr. Murchison did me the honour to reproduce it in his book on 'Diseases of the Liver,' published in 1874, almost verbatim, though setting it up in paragraphs, instead of, as I did, in a tabular form, without mentioning my name in connection with it, I am reluctantly compelled, in mere self-defence, for fear of

being suspected of plagiarism by those who never saw my original monograph on 'Jaundice'—a book which has for many long years been out of print—to draw attention to the fact that my synoptical pathological table of jaundice was published eleven years before it was reproduced by Dr. Murchison. Perhaps, and not at all unlikely, some of my readers, not versed in the pathological history of jaundice, may regard this as quite an uncalled-for piece of reclamation, from their being ignorant of the fact that at the time I drew up the table the whole subject of the pathology of jaundice was nothing more than an unintelligible chaotic mass of theoretical pathological confusion. So that, although the synoptical table may now appear very simple on paper, it was in reality the product of long and deep clinical and pathological study. And just as it is said that Columbus was nearly as proud of having been the first to perform the feat of making an egg stand upon its end (which seems such a trifling matter when we know how to do it), as he was to be considered the discoverer of America, I think that I, in my small way, may be pardoned for feeling anxious to have the correct parentage assigned to my own mental offspring. It being a well-known fact that literary men are, as a rule, as reluctant to lose the credit of the product of their brains as mechanicians are to lose the produce of their hands.

Tabular View of the Pathology of Jaundice, according to the Author's Views.

- **Jaundice**
  - **From Suppression**
    - Enervation . . . . :
      - Fright
      - Anxiety
      - Sudden Joy
      - Over Mental Exertion
      - Concussion of Brain
    - Congestion of Liver
      - Active:
        - Hepatitis from Direct Violence
        - Indigestion
        - Ague
        - Typhus
        - Typhoid
        - Scarlatina
        - Pyæmia
        - Yellow Fever
        - Poison, such as that of Snakes, Phosphorus, Copper, &c.
      - Passive:
        - Heart-Disease
        - Pneumonia
        - Pleurisy
        - Imperfect Respiration in the Newborn
    - Absence of Secreting Substance:
      - Cancer
      - Cirrhosis
      - Fatty Degeneration
      - Amyloid do.
      - Atrophy: Acute / Chronic
  - **From Obstruction**
    - Congenital Deficiency of Ducts:
      - Small Ducts (?)
      - Hepatic Duct
      - Common Duct
    - Accidental Obstruction in Course of Duct:
      - Inspissated Bile
      - Gall-stones
      - Hydatids, and other Forms of Entozoa
      - Foreign Bodies from Intestines
    - Closure of Duct:
      - Stricture
      - Catarrh
      - Pressure of Pregnant Uterus
      - Tumours of the Liver and Gall-bladder, Stomach, Ovaries, and Intestines
      - Impacted Fæces in Transverse Colon
      - Organic Disease of Pancreas, or of neighbouring Glandular or other Organs
      - Abscess in Head of Pancreas
      - Cicatrised Ulcer of Duodenum

The annexed table has the advantage of placing before the reader all the causes of jaundice, as dealt with in the text, in so sharply defined a manner as to render its teachings devoid of all possible traces of ambiguity, so that it requires from me no explanatory remarks for its easy comprehension by any one who has carefully perused the preceding chapters.

We now come to the consideration of a class of

Diseases of the Liver not necessarily associated with Jaundice.

This is a much wider and more important branch of our subject than is commonly supposed. For under the above heading are not alone included a great variety of widely differing pathological conditions of the hepatic tissues; but at the same time embraced within this heading's area are several of the most formidable as well as a few of the most benign of liver affections. It includes, for example, abscess, cancer, hydatids, cysts, syphilitic, fibroid, fatty, amyloid, and several other less common forms of hepatic tissue degeneration and adventitious tissue growths. Each one of the above-named forms of hepatic disease will now be separately considered.

CHAPTER XVII.

ABSCESS OF THE LIVER.

On clinical grounds the subject of hepatic abscess requires to be subdivided into three distinct parts, and each considered separately, notwithstanding that their symptomatology, as well as their pathology, is nearly identical. The subdivisions I make are :—

1. Idiopathic Hepatic Abscesses (Of tropical and of temperate zones).
2. Traumatic „ „
3. Metastatic „ „ (Including the pyæmic variety).

Suppuration of the liver may occur from a great variety of exciting causes, in any country or climate, as well as at any period of life, between early infancy and advanced age.

The Etiology of Hepatic Abscesses.

Until a few years ago it was, indeed I might almost say that it still is, a prevalent notion that all idiopathic abscesses of the liver are, *par excellence*, indigenous forms of tropical disease, and that rarely, if ever, is an idiopathic hepatic abscess to be met

with in temperate climates, except among persons who have previously resided in the tropics. This was the notion I imbibed from my teachers as well as from my text-books, and as a natural consequence it was the one with which I started on my medical career, and one too from which, I candidly admit, I found it hard to emancipate myself. So that I am not in the smallest degree surprised at finding the majority of medical men, with whom I come in contact about liver cases, still entertain the idea that abscess of the liver is *par excellence*, I might even say, a purely tropical disease.

Education and experience have taught me not alone to regard this opinion as an erroneous one, but likewise to regard the very name of 'idiopathic,' when applied to any form whatever of abscess of the liver, as being a most objectionable distinguishing title, from the simple fact that it conveys to the mind a false idea of the etiology of the suppuration. The term 'idiopathic,' being supposed to denote that the disease to which it is prefixed arises without any known exciting cause, is quite inapplicable to suppuration of the liver ; for, as I shall presently, I hope, be in a position to show, every form of hepatic abscess is invariably preceded by a recognisable exciting cause. The cause being, in most cases, not only apparent to the medical attendant (who knows how to discern it) during the lifetime of the patient,

but demonstrable and tangible at the autopsy. Hence the prefix of 'idiopathic' to an abscess of the liver is, logically speaking, a misnomer. Unfortunately, however, as a distinguishing title is required for the purpose of lucid exposition, and the term 'idiopathic abscess' is already in general use, I feel that it will be better for me to continue its employment than to fabricate a new name; for of different names for the same diseases we have already, alas! a superabundance. It is, however, the name only, and nothing more, that I shall retain, for I shall do my very utmost to assist in demolishing what I consider is both a false and a pernicious clinical theory regarding its nature, namely, the belief that an abscess engendered in the liver of a European residing in a tropical country differs in any way whatever from a similar kind of abscess occurring in the liver of an inhabitant of a temperate zone. At the very outset, therefore, I shall advance the proposition that all the varied forms of hepatic abscesses, and most assuredly they are many, have an absolutely identical pathology, no matter in what part of the globe they occur, and that for all clinical purposes they may be both logically and scientifically embodied in two classes—primary and secondary hepatic abscesses :—

1st, Abscesses essentially primarily local, including the two forms of idiopathic and traumatic.

2nd, Abscesses essentially secondary, including

the two forms respectively named metastatic and pyæmic.

For clinical purposes it matters not one iota how or in what order these classes of hepatic abscess are considered ; for every abscess of the liver is nothing but an abscess, whether it be idiopathic, traumatic, metastatic, or pyæmic, in so far as its morbid anatomy is concerned. So I shall first give the general symptoms of hepatic abscesses, and then allude to the defining characteristics of each kind separately.

To begin with, I must state most emphatically that no form of abscess whatever is necessarily associated with jaundice, and that when jaundice accompanies suppuration of the liver it is the direct result of hepatitis, or of some accidental form of occlusion of the hepatic or common bile-ducts, either produced by the presence of the abscess itself, or by the coexistence of other disease. In the next place I think I may equally emphatically state, although it is directly contrary to accepted notions, that no tropical abscess of the liver ever originates spontaneously, but on the contrary, like the hepatic abscesses of temperate zones (when carefully investigated), is found to be the mere sequel or concomitant of some other form of disease in the liver or some other organ of the body. That is to say, of hepatitis, either of the malarial or idiopathic varieties, impacted

inspissated bile or gall-stones, dysenteric or any other form of intestinal suppurations, or even suppurations in parts of the body distant from and unconnected with the liver. All of these exciting causes will be considered in their appropriate places. Meanwhile I may remark that the exciting cause of idiopathic, or so-called tropical, hepatic abscess has long been, and still is, a fearful pathological bone of contention, not so much, however, among scientific pathologists as among clinical physicians; and although to give the *pros* and *cons* of both sides (or I should rather say of all sides, for there are several) of the question would be a most profitless waste of time and energy, I must not entirely slip over the subject, for it has important diagnostic and therapeutical bearings, which merit special attention. I will therefore proceed to say a few more words on the subject, in so far as it concerns practice.

At the outset I may remark that if, to begin with, we accept it as a pathological aphorism that of the multitudinous and varied exciting causes, hepatitis is the most common producer of hepatic abscess, it is easy to understand why an abscess of the liver is more frequently met with among Europeans resident in the tropics than in the same class of individuals residing at home, without fostering the idea that its pathology has anything peculiar or specific about it, at least sufficiently so to render it desirable

to distinguish it by a special name from abscesses of the liver encountered in temperate zones. To the honour of Dr. George Budd be it said, he was the first to call attention to the fallacy of imagining that abscesses of the liver differ from each other according to climate. He clearly showed that, both as regards its clinical history and pathology, an abscess of the liver is exactly the same in a temperate as in a tropical climate, and that the tropical and the temperate abscesses differ in no respect whatever, except as regards their relative frequency. In fact, I go so far as to believe that idiopathic abscess of the liver is limited to no terrestrial zone whatever, being equally indigenous in all latitudes, and that its pathology is exactly the same at the torrid equator as at the frigid North Pole. Having said thus much on the world-wide indigenous distribution of hepatic abscesses, I willingly admit that they are much more common among natives of temperate climates living in the tropics than elsewhere. But this again, I believe, does not arise so much from the hot temperature of the climate as from the mode of life pursued by Europeans while living in tropical countries. I arrive at this conclusion chiefly from the fact that the natives of hot climates, whose mode of life is entirely different from that of the Europeans living amongst them, are not one whit more liable to be affected with abscesses of the liver than Englishmen residing in Great Britain.

Moreover, the twenty years' experience I have had in the treatment of cases of abscess that have originated in patients while living in the East, as well as of cases of precisely the same nature which have occurred in Englishmen who have never put their feet beyond the confines of the British shores, enables me to say emphatically that the chief exciting causes of hepatic abscesses are gluttony and intemperance, which, in proportion to the habits of life, are far more common in Europeans living in the tropics than in the same class of individuals resident in temperate zones, as I have already pointed out at page 242.

It is to be remembered that the words 'intemperance' and 'gluttony' are here employed as relative terms, ruled and modified by collateral circumstances, as will presently be explained. Meanwhile, as bearing forcibly on this question, I may cite a case which goes far to prove that an idiopathic abscess of the liver may occur from what might be thought to be very insufficient causes, even in a temperate zone. The case I allude to is one entitled 'Lobular Hepatitis terminating in Extensive Suppuration,' and is recorded in the 'Pathological Transactions' (vol. iv. p. 171) as occurring in a woman 20 years of age, who died four months after its onset, and who had never been out of England, in whom the only exciting cause *seemed* to be the living in a close hot room in a damp situation.

In case the reader may think that this is not a sufficiently telling example of so-called tropical disease originating in a temperate zone, I shall present him with another more striking example in the shape of an hepatic abscess associated, as in India, with a true dysentery. And it is all the more remarkable, seeing that not only both affections occurred at the same time in the same patient, living in a temperate zone, but that the patient was a mere child. The case was brought before the Pathological Society on January 18, 1881. It is briefly as follows :—'Dr. Norman Moore showed a specimen of abscess of the liver following dysentery in a child aged $3\frac{1}{2}$, who was admitted into St. Bartholomew's Hospital suffering from diarrhœa and pains in the abdomen, accompanied by high temperature. *Post mortem*, the whole large intestine, including the rectum, showed extensive patches of ulceration. In the right lobe of the liver was an abscess as large as an orange, which had penetrated the diaphragm, and opened into the right lung. There was a second abscess in the left lobe. The child had never been out of London.'

In citing these cases I do not desire it for a single moment to be supposed that I ignore the fact that by far the majority of idiopathic hepatic abscesses met with in England occur in individuals who have during some period of their lives been resident in tropical climates. For my own experience,

as well as information obtained from a variety of other sources, has satisfied me that idiopathic hepatic abscess is by no means a very common indigenous disease in England. Indeed, the records of the *post-mortems* performed in our public hospitals in a measure prove this. For example, in the statistics given by Dr. Norman Moore of the results of the autopsies made at St. Bartholomew's Hospital, it is stated that out of a total of 2,464 examinations, there were only twenty cases of abscess of the liver. That is to say, less than 1 per cent. An interesting fact, too, is that the situation of the abscesses in these indigenous cases is almost identical with what it is in the case of Europeans resident in the tropics. Eleven abscesses having been found in the right, one in the left, and nine in both right and left lobes of the liver.

Although statistics of this kind cannot be gainsayed—as far as they go—I dare not venture to say that they correctly represent the relative proportion of hepatic abscesses occurring in England. For my own experience tells me that, at least among the better classes, abscesses of the liver are far more common than here represented. Of which class, of course, hospital statistics take no cognisance, and consequently their data cannot be regarded as furnishing reliable statistics of the relative proportion of abscesses of the liver to other forms of fatal disease occurring among all classes of the community. I

am no less conscious, however, that generalisations founded upon data furnished by private practice would be equally misleading. More especially in cases like my own, where, from the simple fact of my having made the subject of liver diseases a special study, hosts of cases are drifted, as it were, into my net, which under other circumstances I should never so much as even hear of. Consequently the numbers I come into personal contact with in no way represent the relative frequency of cases of hepatic abscess occurring among the general population. But even when all this is taken into account I still opine that indigenous abscess of the liver is a far more common disease than our hospital statistics would lead us to imagine ; and the reason of this is, I think, not far to seek, seeing that not only the modes of life pursued by the upper and lower classes in England are entirely different, but their forms of diet are even more different still. And, as I shall now proceed to show, these are the two most essential elements everywhere at work—in temperate as well as tropical climates—as the exciting causes of abscess of the liver.

To begin with, I may allude to the not unimportant fact in connection with the solution of the question, that not only in the tropics, but everywhere else, it is found that hepatic abscesses are much more common among men than among women ;

and the reason of this, I believe, is easily explained when we consider the marked difference in their modes of eating and drinking. More especially in the tropics, where the whole of their surroundings may be regarded as abnormal. Consequently, when the functions of the liver (pointed out in the physiological chapter, page 65, more especially that which I have spoken of under the head of the calorifying one) are taken into consideration, one cannot feel in the slightest degree surprised that the liver should be not only the first organ of the body to rebel, but the first to give way and entirely break down under the pressure of adverse circumstances.

(*a*) An increase of external temperature arrests, to a certain extent, the normal hepatic calorifying process; for while external cold increases, external heat proportionally diminishes it. (*b*) The climatically enforced indolent habits of its proprietors in the tropics, in comparison with their accustomed muscular activity at home, reduces still further the necessity for the exercise of the liver's normal functions. (*c*) Notwithstanding that its calorifying functions are reduced, the organ is still supplied, as a rule, with the same amount of highly heat-making nutritive foods and drinks. That is to say, foods and drinks rich in hydrocarbons. While again, partly from the increase of external temperature, and partly from the enforced indolent habits of its proprietors,

the hepatic cells not being called upon to consume the hydrocarbons in the normal manner, and yet being forced to get rid of them, have a great amount not only of unaccustomed, but detrimental, labour thrown upon them, in consequence of which they strike work, and at length completely break down. First, the hepatic tissues become congested; secondly, they become inflamed; and thirdly, they suppurate.

This theory is strongly supported by the well-known fact that the natives of the tropics, who, relatively to their surroundings, live quite as regularly and judiciously as the majority of the natives of temperate zones in their own countries do, manifest no greater tendency to hepatic abscesses while living in their tropical homes than the inhabitants of temperate zones do while living in their cooler climates. It appears to me, then, to be quite as ridiculous to speak of the pathology of a tropical hepatic abscess as a thing specially tropical, as it would be to speak of a tropical sunstroke as being something specifically different in its pathology from a case of sunstroke occurring in Hyde Park. Or even a case of frost-bite occurring in Camden Town (as it did a few years ago) as being different in its morbid anatomical relations from a similar case of frost-bite occurring in Iceland. In fact I believe that an abscess of the liver, exactly like a sunstroke, is precisely the same

in every respect when it occurs in temperate England as in tropical India, except as regards its relative frequency. I shall now, therefore, proceed to consider the symptomatology of hepatic abscesses collectively.

General Symptoms common to all Forms of Hepatic Abscesses.

It may be as well for me, before entering upon the consideration of the symptoms, to remind the reader that the diagnosis of hepatic abscess in the majority of cases is no easy matter, and the reason of this is not far to seek. For, in the first place, there is no well-defined class of *symptoms* which distinctly point out the affection; in the second, the *signs* of suppuration of the tissues of the liver are exceedingly indefinite; while, in the third place, the clinical history of hepatic abscesses is in the majority of instances obscure.

What adds most to the difficulties of the diagnosis is the fact that pain, so usually characteristic of suppurations occurring elsewhere, is a symptom of no pathognomonic importance whatever in a case of abscess of the liver, from the fact that not only may there be great pain with only slight hepatic inflammation, but scarcely any pain with considerable suppuration. While again, the amount of pain induced by an abscess of the liver depends much

more on the seat than on the extent of the suppuration, a small superficial abscess being invariably more painful than a large deep-seated one.

Moreover, additional difficulty is thrown in the way of diagnosis by the fact that the abscess is in general so circumscribed that there is not sufficient hepatic tissue affected to give rise to anything approaching to a suppression of the biliary function; so that there is often neither a yellow skin, pipe-clay-coloured stools, nor saffron-tinted urine to assist us. In fact, beyond a mere icteric tint of the complexion and conjunctivæ, with febrile symptoms and hepatic malaise, there may be no physical signs to guide one to the liver; for in many cases there is not even the slightest enlargement of the organ detectible.

I may further mention that the rigidity of the right rectus muscle (which has been vaunted as a pathognomonic sign), though exceedingly useful when it is present, is unfortunately when absent of no negative importance whatever.

The justness of the preceding remarks is amply verified by the well-known fact that it is no uncommon thing even in the tropics to find an abscess in the liver of Europeans who have died from the effects of some non-hepatic form of disease, during whose lifetime the existence of suppuration of the liver was never so much as suspected. To

these cases has been given the name of 'Latent Hepatic Abscesses,' from its being supposed that (as pus once formed in the liver is never absorbed?) they may have existed for years, having probably originated at a time when, from the mildness of the symptoms, only hepatitis had been suspected.

With these general remarks I shall now point out the special signs and symptoms which are usually considered as evidence of suppuration in the liver.

After the ordinary signs and symptoms of hepatitis have been present for some days, the patient suddenly complains of chilliness, rapidly developing into a distinct rigor, which in its turn is speedily followed by an aggravation of all the local signs and general symptoms of hepatitis. The occurrence of the rigor in the course of a hepatitis, though the most reliable sign to go upon, is not, however, an infallible one, from the simple fact that rigors occur in other forms of painful liver disease. Gall-stones, inspissated bile, and entozoa impacted in the ducts, for example, cause rigors, and that, too, unfortunately, occasionally without inducing jaundice. However, when rigors occur in the course of hepatitis without much pain, and they are immediately followed by an increase of constitutional disturbance, the pulse becoming more frequent and hard, the skin hotter and drier, the tongue more

foul, the appetite entirely lost, and great thirst complained of, the uneasiness in the region of the liver being at the same time increased, the tenderness on firm pressure becoming more acute, deep inspiration, sneezing, or coughing causing sharp pain, and lying on the right side being almost next to impossible, suppuration may be diagnosed.

Occasionally, though not often, suppuration of the liver is accompanied by both vomiting and purging; but neither of these can be regarded as a diagnostic sign. Sometimes the formation of purulent matter is exceedingly insidious in its course, and the signs and symptoms of its occurrence are so slightly marked as to altogether escape the notice of the medical attendant. This is more particularly the case when the suppuration follows in the wake of some well-marked form of chronic hepatic disease, such as impacted inspissated bile, gall-stones, or any of the other liver derangements which induce some one of the multitudinous forms of jaundice. In those cases even where there are but slight hepatic pain, little malaise, and only trifling febricula, the supervention of rigors (without any other apparent assignable cause) ought always to raise the suspicion of the occurrence of suppuration.

I must here call particular attention to what is designated the red raw-flesh looking tongue, which some writers have gone so far as to say is a patho-

gnomonic sign of abscess of the liver. This is undoubtedly a mistake; for not only is a raw-flesh looking tongue to be met with in other forms of hepatic disease besides suppuration, even in hepatic diabetes for example, but it is not unfrequently met with in severe forms of gastric derangement quite unconnected with liver disease.

As an addition to our slender means of diagnosis I may mention that in a clinical lecture, published by Deputy Surgeon-General Dr. Furnell ('Indian Med. Gazette,' January 1881), on Abscess of the Liver, he says that he believes that the thermometer furnishes the only reliable guide to diagnosis. But unfortunately the evidence he adduces in support of this opinion can scarcely be said to bear it out; for all that it in reality proves is that the variations of the temperature in cases of abscess of the liver resemble those usually met with in cases of chronic phthisis. My own observations lead me to put down the average temperature at between 102° and 105° Fahr.

Seeing that the diagnosis of hepatic abscess is so perplexing, it may perhaps be as well for me to tabulate the signs and symptoms upon which I myself put most reliance when investigating a doubtful case. They are the following:—

1. The fact of the rigors occurring without any other assignable cause, and being unassociated with paroxysmal pain. For if the pain is paroxysmal it

indicates that the rigors are due to impacted biliary concretions, entozoa, or some other obstructive cause.

2. The co-existence of fever with hectic exacerbations, assuming more of a typhoid than of an aguish character.

3. Intense prostration of strength, associated with gastric derangement.

4. The antecedent existence of some one or other of the many possible exciting causes of hepatic abscess, such as dysentery, biliary concretions, intestinal ulcers, suppurating wounds, either external or internal, even a gonorrhœa, or having swallowed a fish-bone or a pin (see illustrative cases further on).

5. Abscess of the liver is often associated with one or more abscesses in other organs of the body. Even an abscess in the brain, as well as in the spleen, having been found in the same patient associated with an abscess of the liver (vide a case reported by Dr. Moxon in the Pathological Society's 'Transactions,' 1868).

6. An abscess may even exist in a cirrhosed liver, and that, too, along with well-marked jaundice. The abscess in such a case is usually of small size, not bigger than a walnut, or at most an orange, and is in general the result of metastasis.

7. When there is enlargement of the liver, it ceases to be uniform so soon as sufficient matter has formed to cause a bulging of its tissues; and when the abscess

is situated at its lower surface, or when it projects from under the false ribs, it can usually be detected with the fingers as a tense, smooth, tender globular tumour, with a feeling of more or less distinct fluctuation about it.

8. When the abscess is small, and even, I may say, when tolerably large, if seated within the margin of the ribs, palpation furnishes no signs whatever of its existence. For even on the application of firm pressure no pain, beyond perhaps a feeling of diffused uneasiness, is complained of.

9. When the peri-hepatitis which accompanies an abscess of the liver is acute, and aggravated during deep inspiration, it is apt to be mistaken for pleurisy, and all the more likely so in inexperienced hands, when, on the application of the stethoscope, an hepatic friction sound is audible, which is of general occurrence in such cases.

10. A superficial suppurating hydatid is not unlikely to be taken for a case of idiopathic abscess; but when this mistake occurs it is of no moment, for the suppurating hydatid assumes, at least for the time being, all the characteristics and effects of an ordinary hepatic abscess, and therefore demands the same line of treatment. A precisely similar remark may be made regarding a suppurating gall-bladder, which has more than once been mistaken for an hepatic abscess bulging downwards from the liver.

11. The more superficial an hepatic abscess is, the more decided and acute is the pain which it causes. Hence, whenever a change in the character of the pain takes place in the course of a correctly diagnosed abscess—the dull subacute suddenly changing to a sharp acute pain—it may be regarded as a trustworthy indication that the matter is pointing at some part or another towards the surface of the liver, and consequently be looked upon as a favourable symptom. For in any case it at least renders it highly probable that the exact situation of the pointing part will sooner or later become manifest, and thereby the operation of tapping be made comparatively easy and dangerless.

12. Occasionally the diagnosis of hepatic abscess is simple in consequence of the formation of pus rapidly supervening upon the rigors, the collection almost at once pointing at the lower edge of the false ribs, and making its presence visibly apparent to the eye. Under these circumstances the sooner surgical aid is had recourse to, in the majority of instances, the better are the chances of the patient's recovery.

13. Abscesses of the liver often burst spontaneously, evacuating themselves sometimes into the pleural cavity, sometimes into the pericardium, sometimes into the peritoneum, sometimes into the intestines, sometimes into the pelvis of the kidney, and sometimes, though rarely, externally. The rupture

of the abscess into the intestinal canal is its most favourable mode of bursting, yet it seems to be at the same time almost the rarest course it pursues; for on combining the statistics of Waring and Morehead on hepatic abscesses of the tropics, I find that out of a grand total of 424 patients only twelve cases are mentioned in which the abscess burst into the digestive canal.

14. It is well to bear in mind that an abscess may burst into a blood-vessel and prove suddenly fatal. Also that an aneurism of the hepatic artery has been found occupying the cavity of an hepatic abscess. While a still more extraordinary case has been recorded by Dr. Pearson Irving, where an hepatic abscess perforated its way into the stomach, and carried along with it an aneurism of the hepatic artery. ('Path. Soc. Trans.' vol. xxix. p. 128.)

15. When the abscess bursts into the pericardium, it is, as might be expected, rapidly fatal. A typical case of this kind occurred in a man aged 29, who died in the City of London Hospital for Diseases of the Chest, under Dr. Bentley's care. At the autopsy the pericardium was found to be enormously distended, containing no less than half a gallon of sero-purulent fluid, and communicating with a very large abscess in the left lobe of the liver. The liver itself extending to the umbilicus.

16. Occasionally, though very rarely, an hepatic

abscess bursts at different times in two totally different directions. I shall presently relate a case of this kind which fell under my own observation, where the abscess burst first into the intestines, and the opening healed up, and some months afterwards burst into the lung and rapidly killed the patient by suffocation.

17. The last, though certainly not the least important, adjunct which we have at our disposal in settling the question of the presence or absence of pus in a doubtful case, is the exploring needle. An instrument, in my opinion, far too little used as an aid to diagnosis in liver cases, from the mistaken notion that its employment may be attended with disagreeable consequences. For it is a strange fact that a diseased liver may be punctured in more than half a dozen places, not only without detriment, but with actual benefit. In proof that this is no exaggerated statement, I have simply to refer to the letter published in the *Lancet* of August 8, 1863, by Deputy Inspector-General Dr. J. C. Cameron, in which it is recorded that an enlarged liver resumed its natural size after having been freely punctured in the vain search for an abscess, not a drop of purulent matter having been anywhere found.

Before I proceed to cite cases illustrative of the value and importance of the preceding remarks, I shall add a few words on

The Differential Diagnosis of Hepatic Abscesses.

As regards the distinguishing features of the different kinds of abscess of the liver, it is to be specially noted that while the idiopathic and traumatic forms of suppuration are usually single, circumscribed, and of large size, those of metastatic and pyæmic origin are in general not only of small size, but multiple in number. An idiopathic hepatic abscess is usually deep-seated in the tissues of the right lobe of the liver, though occasionally it is superficial, in which case it is in general situated in the diaphragmatic surface of the organ.

Having said that the idiopathic and traumatic abscesses are usually of large size, and the metastatic and pyæmic forms only of small dimensions, I may as well add that while the latter rarely exceed the size of an orange, those of the former variety are sometimes capacious enough to contain more than a gallon of purulent matter. Indeed cases have been met with where the whole of the liver has been transformed into a single great suppurating mass, its capsule merely acting the part of a containing sac to the purulent contents.

A curious case is related by Flint, in his 'Practice of Medicine,' where, in spite of the whole of the right lobe being transformed into a purulent sac, from which after death no less than two and a quarter gallons of

pus were removed by Dr. Rothrop, the left lobe contained no purulent matter whatever.

All idiopathic abscesses, even those of true tropical origin, are not large. Some have been found not bigger than a walnut, and, unless they have been superficial and pointed externally, their existence has only been discovered after death. Indeed the only symptoms of small and deep-seated abscesses may be an indefinite liver pain, slight febricula, and a trifling chill or rigor or two.

The following are illustrative cases, and to begin with I shall cite a most remarkable one both as regards its duration and clinical history, as it is not only an excellent example of an idiopathic abscess in a London resident, but shows that the matter may be spontaneously discharged in two different ways at different times. First, by bursting in the most salutary way into the intestinal canal, and the opening healing up, and months afterwards bursting into the lung and proving rapidly fatal. The case is briefly as follows :—

A City merchant, of middle age, when first brought to me by his medical attendant (Mr. Cresswell), complained of hepatic pain, feverishness, biliousness, and sickness. The symptoms being so decided, and there being no distinct evidence of enlargement of the liver, the case was diagnosed as one of hepatitis tending to suppuration, and was prescribed for ac-

cordingly. After a few weeks' energetic treatment the patient got well, and I saw nothing more of him for about two years, when he returned to me with a note from Mr. Cresswell asking me to examine, prescribe, and report. This second attack was diagnosed as one of subacute hepatitis, and prescribed for accordingly. Hot fomentations, local blisters, and mild mercurials. He again got apparently quite well, and I heard nothing further of the case for nearly two years, that is to say four years after the suspicion of suppuration had arisen. He was now in London, having taken lodgings by Mr. Cresswell's advice, in order to be under my immediate care. On examination the liver was found to be excessively tender to pressure. Percussion he could scarcely bear ; but by careful manipulation the organ was ascertained to have a dull area of six inches in the perpendicular line, and to extend beyond the left margin of the xiphoid cartilage. The skin was sallow, hot and dry, with an afternoon temperature of 102·6° Fahr. The tongue intensely furred. The urine scanty, high-coloured, and turbid with urates. He had night hectic, and complained of having for several days (ten or more) had occasional shiverings. This train of signs and symptoms, with the history of the case, left no doubt upon my mind that suppuration had actually taken place, and consequently a soothing line of treatment, favourable to abscess

pointing, was had recourse to. More especially with the hope that, by the constant application of large hot poultices over the painful hepatic area, the abscess might be coaxed into making its exact whereabouts detectible. On the ninth day after this treatment was commenced, I was agreeably surprised to find that a marked alleviation of all the signs and symptoms had suddenly taken place after a copious stool, described to me as consisting of a pale yellow creamy-looking matter. The abscess was therefore thought to have burst into the intestines, and evacuated itself, or was, I should rather say, still spontaneously doing so, for with a subsequent motion came away more yellow matter.

Nothing apparently could be more fortunate, and in order to favour the evacuation of the purulent matter a teaspoonful of castor-oil, followed by copious draughts of hot gruel, was ordered to be given night and morning. The liver to be frequently fomented with hot water, and gentle though firm pressure to be applied to it, in order to favour, if possible, the exit of the pus. While in the intervals of fomentation the hot poultices were to be continued. Instructions were at the same time given that everything that passed from the intestines should be carefully preserved for my inspection—the first and second motions containing the yellow creamy evacuation having been thrown away.

I may mention that the diagnosis I arrived at in the above case was, I have no doubt, greatly facilitated by the knowledge I had of the results of a *post-mortem* examination of a man of middle age to whom Dr. Sibson had called my attention in St. Mary's Hospital some years previously. The patient, when I saw him, had a distinctly painful swelling in the right hypochondrium. There was no distinct evidence of fluctuation, but a sort of half fluid, half solid feeling, more, as Dr. Sibson said, like a mass of soft putty than anything else, which the patient declared was very painful on pressure. A few days after I saw him he suddenly passed a large purulent-looking motion, after which he had, like my patient, great relief. The swelling also subsided, and his health greatly improved. The improvement was, however, much more temporary than in the case of my patient; for shortly afterwards, and before he was sufficiently well to leave the hospital, he became rapidly worse, and died about a month after the abscess burst into the bowel. On *post-mortem* examination the perforation was found to be at the bend formed by the ascending and transverse colon.

On my next visit to my patient, which was made on the following day, I was greatly disappointed to find that the night-stool contained such an excess of brown watery fæcal matter that the detection of pus in it, by the naked eye, was utterly impossible.

Day by day the stools were looked at, but with entirely negative results. Until, on the seventh day, a copious repetition of the yellow thick creamy stool was announced, which, on examination proving to be almost entirely free from fæcal matter, was at once recognised as true pus both by the eye and the nose. From this time all distressing symptoms subsided, and after a fourth spontaneous, though less copious, evacuation of the abscess (which occurred within the next few days), the improvement in the condition of the patient was rapid—so rapid that within a month he was able to leave town, comparatively speaking, quite convalescent.

The next time I saw this patient was about seven months later, along with Dr. Macleod at Ben Rhydding in Yorkshire, whither he had gone for change of air. On examining the liver I found that it had again increased in size, and to the naked eye there was a marked fulness in the right hypochondriac region, with an indefinite obscure sense of elasticity over the liver immediately underneath the margin of the false ribs, occupying an indistinct area of about three or four inches in lateral as well as perpendicular diameter. Beyond which, and tenderness on pressure, there were no other signs of a purulent collection in the tissue of the liver. The tongue was moderately clean. The temperature of the skin almost if not quite normal. Appetite natural. Bowels regular, and the pulse

ranging, I was told, from 88 to 94. The condition was now diagnosed as one of chronic hepatic abscess. So I advised artificial evacuation, but neither Dr. Macleod nor the patient seconding the proposition, on account, as they said, of there being no urgent symptoms, a strong iodine liniment and warm fomentations were ordered to be applied over the supposed seat of the abscess, in the hope of getting it to point externally. At the same time, to prevent further suppuration and to favour absorption—if that were possible—a mixture containing quinine and iodide of potassium was to be given thrice a day, and the patient's strength to be supported by plenty of nourishing non-stimulating food. Fresh air and carriage exercise were also recommended.

Some months afterwards I was again summoned to the gentleman's bedside, and I now found a sad change had taken place for the worse. The local symptoms, though but slightly exaggerated, had induced grave constitutional disturbance. Fever had been succeeded by marked hectic, hectic by pyæmia, the pyæmia being associated with considerable derangement of the cerebral functions, rapidly running on to delirium. As the patient was then residing in the Bloomsbury district, I put him under the immediate care of Mr. William Gill, a gentleman in whose professional skill I had entire confidence, and who, from the proximity of his residence, could not only

easily see him frequently, but, on an emergency, almost at a moment's notice.

The condition of the patient daily became worse. Until at length the abscess suddenly burst, and poured its contents into the right lung. And the poor patient, not possessing enough physical strength to enable him to expel sufficiently rapidly the purulent discharge from the bronchi by coughing, died asphyxiated in the course of a few minutes. This was a most instructive case to me. Not only from my having it under observation for more than five years, but on account of its having presented at various times so many different phases in its clinical history. Not the least among its peculiarities may be reckoned the long period—nearly six years—of the hepatic abscess's existence.

The friends were sufficiently enlightened and liberal to offer me the privilege of making a post-mortem ; but unfortunately I was too much professionally occupied at the time to be able to avail myself of it, and, Mr. Gill not caring to make the autopsy without my presence, the opportunity was let slip. A circumstance which I have since deeply regretted for many reasons, but for one in particular. Namely, that I had observed that the night-dress and bed-sheets, on the morning of the patient's death, were deeply stained of a bright vermilion colour, from something that came away along with

an involuntary motion passed during the act of dying. At first I thought that it might be Condy's fluid, or some logwood, or other red-coloured disinfectant or medicine; but, as my enquiries failed to elicit the slightest clue to the origin of this colouring matter, I am forced to the conclusion that it was some highly oxidised animal pigment, which had been expelled along with the fæces during the agony of death. This peculiar staining of the night-dress and bed-sheets has now to me a very special interest from the fact of Mr. Thomas Pick having communicated a case to the Pathological Society in 1869, where in the interior of an enormous hydatid of the liver (from which before death thirty-one pints of a dark greenish albuminous fluid, containing quantities of cholesterin, but no echinococci) were found a number of masses of the size of horse-beans, consisting of a 'material exactly resembling vermilion'! Which, upon microscopic examination, were discovered to consist of rhomboidal crystals of hæmatoidin (Fig. 29). Probably an autopsy in my case might have thrown some light upon the source of the red colouring matters which are every now and again observed to pass away by the stools in cases of hepatic abscess, and which (as in a case presently to be referred to) have been described as bearing a close resemblance to 'red-currant jelly.' They are not uncommon in hydatids.

Biliary Concretions as a Cause of Hepatic Abscess.

I shall now cite a few cases in proof of the statement that biliary concretions, both inspissated bile and gall-stones, induce abscesses of the liver. The first case I shall refer to is one reported in the fifth volume of the Pathological Society's 'Transactions,' p. 161, by Dr. J. W. Ogle. The patient, a man of middle age, died immediately after entering St. George's Hospital. The skin was jaundiced. An abscess was found in the right lobe of the liver, full of dark offensive fluid in which were a number of variously sized polygon-shaped biliary concretions, agglutinated together with mucus. The mass, which was the size of a small hen's egg, was supposed, from its shape, to have been originally in the gall-bladder, not a trace of which, however, remained. Dr. Ogle's theory was that ulceration of the gall-bladder had set up inflammation, which had spread to the neighbouring surface of the liver and ended in the formation of an abscess, into which the gall-bladder, with its contents, ultimately merged. A case of abscess in the left lobe of the liver induced by a gall-stone impacted in the common bile-duct in a woman aged 23, who died in St. Bartholomew's Hospital, will be found in the twenty-fifth volume of the Pathological Society's 'Transactions,' p. 133.

The following case of abscess induced by gall-

stones is one which came under my notice in 1868. It was that of a barrister who, after having been for a few years in the East Indies, returned home suffering from liver disease. He was 44 years of age, and said he had been attacked with excruciating pain in the liver while in India. Which was diagnosed by his medical attendant as the effects of entozoa. On carefully examining his liver, I found it enlarged and tender on pressure, more especially in the region of the gall-bladder. On getting him to describe to me minutely his symptoms, I perceived that the pain had been caused by gall-stones and not entozoa, and that he had had an attack of acute hepatitis consequent thereon. After a few months' sojourn in England, the question of the propriety of his returning to his Indian practice arose, and I, knowing well the constitution of the man I had to deal with (he being a friend of several years' standing), at once told him that to think of returning to India under the circumstances would be an act of madness. For most assuredly before he was many months back in a hot climate he would have another, and most probably a much more severe, attack of acute hepatitis. From which he would either die there, or merely return home to die, as a liver in the condition his then was could not possibly stand the mode of life in a tropical climate with impunity. He then asked me why I took such an unfavourable view of his case, seeing

that he had no urgent symptoms. This led me to review to him his clinical history, and I particularly dwelt on the fact that, although he had no pain now, and even but very little discomfort, his liver was nevertheless not only congested, but chronically enlarged. That there was a distinct fulness and tenderness in the region of the gall-bladder, which I believed was due to the presence of one or more gall-stones. Adding, moreover, that he knew as well as I did that he had a marked constitutional tendency to biliary derangements, although not cognisant of the fact that it took the form of biliary concretions. When all these facts were taken together, in conjunction with his already generally enfeebled state of body, my experience of the effects of a life in tropical climates in similar cases was such as led me to the belief that his return to India would not only be followed by another attack of acute inflammation of the liver, but most probably a severe one, which in all likelihood would run on to suppuration, and, if it did so, would most certainly kill him. On my putting the matter thus clearly and emphatically before him, he at once said: 'I believe you are right. I won't go back to India.' But alas! poor man, he had a wife who, I suppose, found an Indian life better suited to her tastes than an English one. At any rate she apparently had made up her mind that back to India they should go. Consequently I received a visit from

her, the object of which was—I won't say to make me change my mind, but I will say—to change the wording of my opinion. All that she wanted, she said, was for me merely to give my consent that her husband should be allowed to go back to India for a month or two on trial. Not finding me so amenable to persuasion as she had anticipated, however, and being not only determined to get her own way, but, what was more, to shelter herself from the censure of her husband's friends under the cloak of a 'doctor's opinion,' she took him back to an eminent consultant who had seen him on one or two occasions after his return from India. What took place at that interview I know not. All I know is that she assured me that he had said that 'there was not the slightest danger in her husband's returning to India.'

The fatal step was soon taken. Back to India he went, and no sooner was he there than he had a severe attack of hepatitis which made him return almost immediately to England. But alas! too late. For suppuration of the liver had already set in, exactly as had been prophesied; and the accompanying extract from a letter written to me by Mr. Sibley, who attended him in his last illness, tells the sad end of one who, had he but had a little more control over his own actions, would, in all probability, be at this moment still alive and well. Mr. Sibley says in his letter: 'When I commenced to attend him, his symp-

toms were urgent. There was a considerable amount of fever, at uncertain times. The abscess was opened by Mr. De Morgan, and, as a large quantity of pus was found, an ordinary drainage-tube was left in. He seemingly did well for several days ; but symptoms of exhaustion set in, and he died on the fourteenth day after the opening of the abscess.'

Let this sad history be a warning to the reader never, under any circumstances whatever, to advise a patient with gall-stones, or who has had even a single attack of acute hepatitis, if he has a constitutional tendency to biliary derangements, to reside in a tropical climate. For, even under the most favourable circumstances, the probabilities are that not only will his hepatic functions rapidly get out of order, but in all likelihood he will contract some fatal form of liver disease. It is always best to err on the safe side ; and even when there are strong pecuniary inducements for disregarding the above advice, the patient should be made thoroughly to understand the risk he runs, and the responsibility be thrown entirely on his own shoulders.

Abscess of the Liver from Embolism.

The following case of hepatic abscess, supposed to be the result of embolism, was brought before the Pathological Society (February 1881) by Dr. Andrew Clark.

The patient, aged 40, never suffered from any illness until June 1880. About that time, after leaving Calcutta, he began to feel ill, to lose flesh, and was feverish at times. He landed at Natal, and there was told that he had an enlarged liver. Not improving, he pursued his journey to England, and soon after his arrival was admitted into the London Hospital. When first seen, there were no physical signs of disease in any organ, beyond that the liver appeared to be smaller than natural; there were no tender spots; he occasionally suffered from night sweats. An aspirating needle was introduced from right to left through the organ; after some little difficulty, about ten ounces of reddish-yellow matter were drawn off. For about a week after, physical examination revealed that the liver reached upward in front as high as the nipple, and a little higher behind; over the liver a dry friction-rub was heard; and the right lung was consolidated as high as the middle of the scapula. The patient died a few days later; a short time before his death, he coughed up some pellets of the colour of the matter drawn off at the aspiration; elastic tissue from the lung was found in these pellets, and also evidence of bile-pigment. *Post mortem*, an oval abscess was found in the middle and back part of the liver, reaching as high as the level of the root of the lung; in the liver-tissue around it were numerous

smaller abscesses. The large intestine showed numerous ulcers, extending from the caput cæci to the hepatic flexure. Dr. Clark thought the origin of the abscess was probably embolic. I myself regard it as being due to the presence of the intestinal ulcerations. I, however, record it as a case of abscess from embolism, in order that further investigation may be excited by its narration.

As I have more to say regarding the causes of so-called idiopathic abscesses, which, as I said before, in no way differ in their pathology from many of those which are denominated metastatic abscesses, I shall, in order to prevent repetition, delay their further consideration until I come to the exposition of the metastatic variety.

Traumatic Hepatic Abscess.

Abscesses of the liver as the result of external violence are exceedingly rare, and when they do occur are in general the result of a blow or of a squeeze.

Traumatic suppuration of the liver may occur at any age. It has been frequently met with in children. A case of this kind in a little girl, aged 9, came under the care of Mr. Obré. She fell downstairs, and hurt her side, and three or four months afterwards there was noticed a distinct intercostal bulging at the seat of the injury, which proved to

be an abscess. For on an exploring needle being inserted at the junction of the epigastric and hypochondriac regions, about a pint of pus was evacuated. A second evacuation was made a few days later, but the child sank and died. At the *post-mortem* a large hepatic abscess was found in the right lobe, containing about a pint and a half of greenish-brown thick pus. The gall-bladder was contracted and healthy. The hepatic tissue round the abscess presented an inflamed, deep red hue, and was hardened, as is usually the case in inflamed hepatic tissues. No ulceration of the intestines was anywhere to be seen; so the abscess was put down as the direct result of the injury which the child had met with four months before.

Sometimes, indeed, even a succession of abscesses form in the liver after severe mechanical injury. A very curious case of this kind, where an abscess of the liver, which burst into the peritoneal cavity fourteen days after the receipt of the injury, was followed by the formation of a second abscess (a little away from the first), which in its turn burst, eleven days later, into the intestines, occurred in a member of our own profession, aged 69, who was thrown from and injured by the upsetting of his vehicle. It is recorded in the fourth volume of the Clinical Society's 'Transactions,' by Dr. John Harley. The case is peculiarly interesting, so I shall quote it

at some length, as not the least remarkable feature in it is the fact that recovery took place after the second abscess had continued to discharge itself more or less interruptedly during three months.

The salient points in the history of the case are that although the gentleman was able to ride home on horseback immediately after the receipt of the injury (which he at first merely spoke of as a bruise in the epigastrium), on the seventh day he was suddenly seized with a deep-seated pain in the upper part of the right side of the abdomen, accompanied with vomiting. In a few days more there was an elevation of the right rectus muscle, where the finger could detect a solid deep-seated tumour, continuous above with the liver. It was exquisitely tender to the touch, and painful on coughing. The pulse was 80 ; the tongue clean and moist. There was no thirst. The urine was high-coloured ; the bowels regular. On the fourteenth day, when turning in bed, he was suddenly seized with agonising pain in the epigastrium, and became bathed in a cold sweat. The abdomen was tense, and everywhere tender to the touch ; but 'the special pain, dulness, and tumidity on the right side had disappeared.' 'The symptoms indicated, and subsequent examination proved, that the tumour had ruptured and its contents been diffused.' Dover's powder was given, and a tranquil though sleepless

night was obtained. On the following day the pulse was 120. No tumour could now be seen or felt. The urine was scanty, of an orange tint, and deposited lithates. He perspired freely. He lay easiest on his back, with thighs outspread and legs flexed. At night, again a sudden change occurred. After turning in bed, he became faint, and bathed in cold perspiration—pulse 160—and was in a state of collapse, which lasted for three hours. He then slowly rallied, but remained prostrate up till the twentieth day, when there began to be a decided improvement in his condition—pulse 96, regular and good. On the twenty-second day he was attacked with pleurisy of the left lower lobe. On this day he passed a motion containing a mass of 'clear red mucus, exactly like red-currant jelly.'

On the twenty-fifth day the pleurisy was gone, but to the left of the umbilicus there was a painful tender fulness, and again another mass of 'tawny red jelly-like mucus' was passed, which was soon followed by an offensive motion—'blood-stained mucus, strings of colourless mucus, and black specks of altered blood.' The swelling now seemed to subside, with a gurgling sound on gentle pressure, and a 'large fluid, almost involuntary, evacuation immediately followed, with much flatus, causing a scalding sensation.' 'It was pus.' The whole motion amounted to about twelve ounces in quantity.

Two hours afterwards there was no tenderness of the abdomen left. Pulse 96. Tongue moist, and like a piece of raw meat. After the next day 'no pus was discharged, but there was a constant oozing of clear watery fluid, faintly tinged with blood.'

On the forty-second day three motions, partly of healthy fæcal matter and partly of offensive purulent matter, were passed with relief. From the forty-ninth until the fifty-first day, 'large quantities of healthy pus were passed, both alone and with the fæces.' On the fifty-fourth day, the jelly-like mucus reappeared. The bowels acted naturally. On the fifty-fifth jaundice appeared, accompanied with great abdominal distension and pain. At noon he began to get relief by the discharge of offensive purulent fluid from the bowels, and it continued to run away involuntarily during the rest of the day. He passed in all about a quart; it was partly ochre-coloured, and partly pure greenish-yellow pus.'

On the fifty-sixth day the jaundice disappeared. He was free from pain, and the stools were natural, but with a little pus. On the 112th day the pus finally disappeared. He was by that time able to walk in his garden, and he soon afterwards regained his weight and usual healthy appearance.

Nothing is said in the report regarding the probable cause of the temporary attack of jaundice, which lasted only a single day; and as there is

nothing in the clinical history of the case which offers any direct clue to its solution, I merely call attention to the fact, in order that in future cases some note may be taken of what might be regarded as a peculiar phenomenon by some, but which I think was due to the accidental presence of a biliary concretion.

Metastatic and Pyæmic Abscesses of the Liver.

Under this heading is included a long and varied series of pathological affections, which, though at first sight apparently incongruous, prove on close examination to be not only nearly allied, but actually identically produced forms of suppuration. Having, as I think, already successfully undermined the hitherto supposed right of any form of liver abscess whatever to the specific title of 'Tropical,' I shall now in like manner attempt to prove that the metastatic hepatic abscess which follows as a sequela to tropical dysentery, in spite of all that has been written to the contrary, is not due to any specific dysenteric poison whatever, but simply and solely to the absorption of pus into the blood from the purulent ulcers in the intestines, just as occurs in the course of the ordinary metastatic and pyæmic abscesses met with in England as sequelæ to intestinal, urethral, and other suppurations. In order to demolish the, as I believe, false pathological doctrine regarding the specific connec-

tion between tropical dysentery and hepatic abscesses, I shall cite a few crucial cases, the careful consideration of which will, I think, of itself be sufficient to scatter to the winds this 'specific' theory which has long obstructed the path of clinical truth.

The first case I shall quote is one brimful of important and incontrovertible data. It fell under my notice when house physician in charge of the fever wards of the Edinburgh Royal Infirmary in 1851, and is briefly as follows.

Pins a Cause of Hepatic Abscesses.

A well-built and well-nourished lad of 19 years of age was sent into the male fever ward by the admitting physician under the impression that he was suffering from typhus fever, and I, not being then as *au fait* at liver cases as I am now, ignorantly treated the case as if it were one of typhus fever with anomalous symptoms. The anomality of the symptoms consisting in the fact that the disease had apparently flown to the liver instead of to the brain. The lad was very ill, his pulse rapid, his skin hot, his tongue foul and tremulous. The liver, though not enlarged, was excessively tender on percussion. There was neither jaundice nor bilious urine, and the stools were of the normal colour. A few days after his admission he was seized with pulmonary symptoms —thought to be pneumonia—rapidly got into a hec-

tic, and then into a low typhoid state, and died nine days after coming into the hospital. A *post-mortem* examination was made, and an unsuspected abscess, the size of a swan's egg, was found embedded in the substance of the right lobe of the liver. Besides two smaller ones, the larger of them about the size of a walnut, were found in the middle lobe of the right lung. On examining the intestines a drop or two of pus was found in the peritoneum, on the outside, and at the very apex, of the appendix vermiformis ; and its cause was soon discovered to be the presence of a thick brass pin, about an inch and a quarter in length, with its head somewhat green and eroded, pointing downwards, and projecting half through the caudal extremity of the appendix vermiformis. Here then is an excellent example showing how an abscess of the liver may occur in an otherwise healthy constitution, through the medium of metastasis, from a drop of purulent matter.

Dr. Payne has recorded a very similar case, where a pin lodged in the vermiform appendix became surrounded by a concretion of fæcal matter about an inch long, which led to the formation of four hepatic abscesses, one of which was large enough to contain ' many ounces of greenish pus.' The patient, a woman aged 37, gave no account of ever having swallowed the pin, and the only clinical history of the case obtainable was that, three weeks before her

death, she was seized with severe pain in her right side, which was more or less continuous, and accompanied with slight tenderness of the abdomen. The bowels were regular, pulse 110, respirations 36, and temperature 104·5°. Four days before her death, she fell into a state of stupor, and a gangrenous patch appeared upon the sacrum the day before she died. At the autopsy it was observed that although the coats of the vermiform appendix were thickened, they showed no signs of inflammation. Yet 'the hepatic abscesses were precisely such as would be called pyæmic,' although 'no seat of primary suppuration was discoverable.' (Pathological Society's 'Transactions,' vol. xxi. p. 232.)

The following is another case of abscess of the liver from perforation of the appendix vermiformis and cæcum by a pin, recorded by T. Whipham, M.B., in the twelfth volume of the Clinical Society's 'Transactions.' It occurred in a lad 18 years of age, who had suffered from severe pain in the right side, extending to the flank; vomited, and been purged during six days at the rate of fourteen motions a day. The right inguinal region was so tender that it was impossible to examine it properly. At the autopsy the liver weighed six pounds. Beneath its under surface was a large abscess containing 'red-brown pus.' A limited abscess was found in the right iliac region, and its purulent contents were

mixed with fæces, which had found their way thither from the cæcum. A pin one and a half inches long was found in the cavity of the abscess.

In none of the above cases was anything known of how the pins found entrance into the body.

Fish-bones inducing Hepatic Abscesses.

In the same way as abscesses of the liver may arise from the irritation caused by pins in the digestive canal, they may follow upon that resulting from the presence of fish-bones. The 'British Medical Journal' of February 26, 1881, says that two cases following the swallowing of fish-bones are recorded in the last number of the 'Nordiskt Mediciniskt Arkiv.' The first case was reported by Dr. Carl Wettergren. The subject, a man aged 41, in September 1875, swallowed a $1\frac{1}{2}$ inch long bone of a bream; at the time, it produced much pain, which soon passed off. In October he had an attack of suppurative perityphlitis, and another in the autumn of 1876. After his recovery from this, he had pain in the course of the transverse colon. On July 29, 1877, he had a severe rigor, followed by deep collapse. He died in October. At the necropsy, the upper third of the vermiform appendix was found to be quite obliterated, while its lower two-thirds were distended into a cyst. There was no trace of perforation. Adhesions were found to

exist between the upper part of the duodenum, the transverse colon, the sigmoid flexure, and the portal region of the liver. The left lobe of the liver contained several abscesses of various ages ; and in one of them, lying close to the suspensory ligament, a fish-bone was found. Dr. Wettergren assumed that it entered the vermiform appendix, then passed between the laminæ of the lesser mesentery, and reached the retro-peritoneal connective tissue, in which it caused inflammation and suppuration. The second case is related by Dr. E. Winge. The subject died after pain in the right hypochondrium, with swelling and tenderness of the liver. At the necropsy, on the upper surface of the liver, and in the organ, numerous small yellowish-white branched bodies were seen ; pus escaped from them on pressure. There were also numerous small abscesses in the peritoneal and mucous membranes of the gall-bladder. A decolorised thrombus, as thick as a thumb, was found in the main trunk of the portal vein ; a fish-bone projected half an inch into the lumen of the vessel ; and, on tracing its course, the other end was found in the jejunum.

A somewhat similar case of this kind, where the presence of a fish-bone in the portal vein produced abscess of the liver, is given by Flint in the fourth edition of his ' Practice of Medicine,' p. 556.

A Stricture of the Rectum may cause an Abscess of the Liver.

Any form of suppurative intestinal disease seems capable of producing hepatic abscesses of a metastatic or pyæmic character. Dr. Wilks exhibited specimens at the Pathological Society, where an abscess, or, I should rather say, a diffused purulent infiltration of the liver, and a gall-bladder filled with purulent bile, were distinctly traceable to the suppuration arising from an ulcerating stricture of the rectum, consisting of dense fibrous tissue, situated about four inches from the anus of a man aged 37. The case is reported in the eleventh volume of the Society's 'Transactions,' and the liver is described as having been found in a state of 'diffused suppuration,' without any separate distinct abscesses in it; but of a uniform condition of purulent infiltration. The gall-bladder was filled with 'a curdy yellow bile.' With the exception of the suppurated liver and the ulcerated and constricted rectum, all the organs of the body were healthy. Suppurating ulcers of common typhoid fever, contrary to what has usually been stated, occasionally give rise to hepatic abscesses. A good example of this kind, where one of the abscesses in the liver contained thirty-seven ounces of pus, was met with in a man aged 40, who died in Guy's Hospital in 1870.

Tuberculous ulcerations of the intestines, on the other hand, as far as I am aware, have never been found to give rise to hepatic abscesses. So that one might almost be inclined to say that there must be something peculiar in the forms of intestinal suppurating ulcerations which give rise to hepatic abscesses. But I very strongly suspect that ere long hepatic abscesses will be met with traceable to pus effused in tuberculous intestinal ulceration, although hitherto none have been recorded.

Gonorrhœa as a cause of Hepatic Abscess.

That an ordinary gonorrhœa may induce suppuration of the liver, has been proved by a case that was published by Dr. Bristowe. It occurred in a shoemaker, aged 50, who died in St. Thomas's Hospital in 1853. He was admitted labouring under 'suppuration of the vesiculæ seminales and prostate,' and on *post-mortem* examination it was found to have induced multiple secondary abscesses in the liver, the lungs, and the kidneys.

Suppurating Hydatids of the Liver give rise to Secondary Hepatic Abscesses.

A case of this kind died in the London Fever Hospital in 1866. The patient, a man aged 35, stated that he had never had a single day's illness until five weeks before his admission, when he was suddenly seized with a pain in the liver, began to

feel sick and to lose his appetite. In three weeks' time he became deeply jaundiced. The stools were loose and pipeclay-coloured, and the urine like old ale. His tongue (on admission) was very red, and the hepatic dulness in the perpendicular right nipple line was eight inches. A distinct, smooth, painless, fluctuating tumour was to be felt, but no hydatid fremitus could be detected in it. A fortnight after admission the patient was seized with rigors, which recurred at irregular intervals for eighteen days, when he sank and died. On *post-mortem* examination a collapsed suppurating hydatid cyst, as big as a child's head, was found in the right lobe, near the posterior margin of its under-surface. No other hydatids were found in the liver, but the entire organ was studded over with small abscesses, varying in size from that of a pea to that of a chestnut. The cause of the jaundice was thought to be the compression of the common bile-duct by the hydatid. Two or three other hydatids, smaller than an orange, were found in the renal region, attached to the peritoneum. In the hepatic hydatid was a quantity of bilious-looking pus, in which floated a number of secondary vesicles. There were no ulcerations of the bowels, or other suppurating sores, detected in the body. Dr. Murchison, under whose care the patient was, attributed the abscesses of the liver to the result of metastasis from the suppurating

hydatid. A fortnight before the patient's death, about six ounces of thin opaque bilious fluid, getting purulent towards the end, were drawn off from the tumour by a small trocar and canula, and at the *post-mortem*, although no sign whatever of inflammatory action marked the course of the trocar, it was seen to have passed through at least an inch and a half of healthy hepatic tissue before it had arrived at the cyst. A consolatory therapeutical observation greatly in favour of hepatic tapping. (Pathological Society's 'Transactions,' vol. xviii. p. 123.)

As suppurating hydatids have induced not only hepatic abscesses but fatal pyæmia, whenever such an unfortunate occurrence as suppuration of an hepatic hydatid is suspected to have taken place, it ought immediately to be punctured with a small exploring trocar, the contents of the cyst emptied, and, when practicable, the cyst itself washed out by the repeated injection of an antiseptic solution. Sometimes suppurating hydatids give rise to what have been described as gangrenous abscesses of the liver, from their contents being excessively fœtid and the surrounding tissues both friable and decomposed. A case of this kind will be found in the Pathological Society's 'Transactions,' vol. xviii. p. 145.

What is the Difference between a Metastatic and a Pyæmic Hepatic Abscess?

The only difference between an ordinary metastatic and a so-called pyæmic hepatic abscess that I know of exists in nothing, unless it be in the relative severity of their signs and symptoms. The pyæmic form usually being the worst, from its occurring in more enfeebled constitutions, and, as a natural consequence, being in general attended with more marked hectic and other febrile symptoms. The most typical case of what might be called pyæmic hepatic abscess that I ever came across, occurred in a middle-aged gentleman whom I saw at Teignmouth, in 1868, in consultation with Dr. Magrath. The most remarkable feature in the case being the severity of the nocturnal exacerbation of hectic, associated with profuse offensive perspiration. There was no jaundice, indeed no icteric tint whatever; which, if it had been present, could not possibly have escaped notice, as the patient was a very decided blond.

The liver was enlarged, and exceedingly tender upon firm pressure, more especially exactly over and all round the neighbourhood of the gall-bladder. The case slowly terminated fatally, and at the autopsy a number of small abscesses were found in the liver, and an agglutinated mass of firm tissue occupied the position of the common bile-duct, which

was in a suppurative condition, in consequence, as was supposed, of the previous irritative effects of an impacted gall-stone, which had ulcerated its way into, and escaped unnoticed by, the intestines.

As the *rationale* of pus-action in the production of secondary abscesses of the liver is as yet unknown, I may here with advantage cite a few passages from a paper by Mr. Watson Cheyne on a 'New Method of arresting a Gonorrhœa' ('British Medical Journal,' July 24, 1880). For having shown that gonorrhœal pus may induce a hepatic abscess, his remarks may probably throw some light on the possible mode of pus metastatic action.

Mr. Cheyne says that 'the extreme contagiousness of gonorrhœa, the existence of a distinct period of incubation, and the steady spread of the inflammation from a given spot, all point strongly to a parasitic origin. Acting on this idea, he made, in the spring of 1879, a number of inoculations of gonorrhœal pus, under certain precautions, into flasks containing infusion of meat or infusion of cucumber. In these flasks micrococci grew in large numbers, and also sometimes bacteria, showing that these organisms were present in the gonorrhœal pus.' Circumstances prevented him from pursuing the subject further at that time. In the meantime, Dr. Neisser published an elaborate research on this subject, in which he showed the presence of enormous

numbers of micrococci in gonorrhœal pus, and in the pus from contagious ophthalmia.

In erysipelas, it has been demonstrated that the skin at the margin of the inflammatory redness is full of micrococci. Koch found, in erysipelas in rabbits, that bacilli were present throughout the inflamed part, and co-extensive with the inflammation. The same writer obtained a progressive gangrene of the tissues in mice by the injection of putrid blood, and he demonstrated conclusively that the gangrene is due to an organism which he calls a streptococcus, which is present in large numbers around the limits of the gangrenous part.

From researches like these, one is led to the conclusion that the production of multiple secondary abscesses may be due to the propagation and spread of living animal or vegetable organisms, and not actually to the pus-corpuscles themselves; and it is this belief, as well as that all the various forms of contagious and epidemic jaundice are the direct result of disease-germs, which induced me to say so much on germicides in the chapter specially devoted to treatment. Figures of the germs are given at p. 335.

Are so-called Tropical Abscesses of the Liver specially due to Dysentery?

This question has long been, and still is, a bone of contention. Some regarding dysentery and abscess as cause and effect; others saying that they are

frequently associated together, simply because they both have similar exciting causes. I think, after careful perusal of the preceding cases the reader will have little difficulty in deciding which side is most likely to be in the right. For if such trivial amounts of pus in the digestive and urethral canals as have been here indicated are of themselves sufficient to induce a series of secondary abscesses in the liver, pleura, lungs, and iliac regions, in what might be otherwise supposed to be perfectly healthy constitutions, in youthful inhabitants of temperate climates, unaccustomed to an excessive indulgence in either rich foods or stimulating drinks, how can one possibly be surprised, or regard it as in the least degree extraordinary, that an equally trifling amount of purulent matter in the intestinal canal in cases of dysenteric ulcerations, &c., should in like manner be sufficient to induce the formation of an abscess in the liver of an Anglo-Indian, with a constitution undermined by malaria, and a liver upset by over eating and drinking, while living in an abnormally high atmospheric temperature favouring all forms of hepatic congestions and inflammations ? Dr. Finlayson, in the 'Glasgow Medical Journal' of February 1873, gives it as his belief that, instead of hepatic abscess being the result of dysentery, the disease of the liver is in reality the cause of the dysentery ; but unfortunately he has adduced no telling facts in support of this bold theory.

In a letter to the 'Lancet' of May 14, 1881, Sir Joseph Fayrer also adds the weight of his opinion to the side of those who believe that statistics do not support the theory that tropical abscesses of the liver are due to dysentery. The statistics he draws this conclusion from being those of Moore in the 'Annals of Military Surgery,' where it is stated that twelve observers in the aggregate report 1,532 cases of dysentery in which only 295 were followed by hepatic abscess, thus giving only 18 per cent.! and as Moore justly remarks, 'if the theory were sound, how could it possibly happen that 77 per cent. of the cases of dysentery occurred without hepatic disease?'

As it would be wrong of me to ignore the views of those who hold opposite opinions, and yet, from my not believing in them,it would be almost equally wrong for me to take up space by detailing them, I beg to refer those of my readers who take special interest in the question to the admirable report on the subject furnished to the Pathological Society in 1858 by Dr. Bristowe (vol. ix. pp. 241–269), on the connection between intestinal ulcerations and hepatic abscess. The conclusion he arrived at being that abscess of the liver cannot be regarded as a consequence of intestinal ulceration; nor dysentery be regarded as the result of hepatic disease, but that a general common cause may excite in one man abscess of the liver, in another dysentery, and in a third a

combination of the two. Although I am adverse to the first of these three theories, I am quite in accord with the last two. I would further recommend the perusal of Dr. Bristowe's paper on the modes in which hepatic abscesses may be formed, at pp. 273–294 of the same volume of the Society's 'Transactions.' Both communications are pregnant with closely reasoned data.

Before leaving the important subject of abscess of the liver, it may, perhaps, be as well for me to state the general conclusions I have arrived at after having given considerable attention to all sides of the question. They are these :—The abscesses which occur in the livers of Europeans living in the tropics (as a sequel to dysentery or to anything else) have precisely the same pathology as abscesses of the liver occurring in persons resident in temperate zones. And they only differ in being more common and at the same time, perhaps as a general rule, more severe in patients who have resided in hot climates. These differences, I believe, are not, however, due to anything whatever specific about the abscess itself, but arise wholly and solely from the habits of the patients while resident in the tropics, together with their surroundings, favouring not alone the production of the severer forms of hepatic congestions and inflammations, but likewise hepatic tissue suppurations. Further, I have been equally

led to the conclusion that the so-called idiopathic abscess of the liver is in the majority of cases actually nothing more or less than a form of metastatic abscess, the *fons et origo* of which has eluded detection. The exciting cause perhaps having been a dysenteric or other form of intestinal suppuration, of a malarial or other origin. For be it remembered that an enlarged liver and spleen, with a tendency to suppuration, are the very commonest of all the sequelæ of malarial poisoning. So common indeed is the disorder of the spleen that one of its forms has been named ague-cake.

Treatment of Hepatic Abscesses.

The treatment of an hepatic abscess, whether it be of the idiopathic, traumatic, metastatic, or pyæmic varieties, in so far as the local suppuration is concerned, is always the same. But the constitutional treatment varies according to the cause of the suppuration. Unfortunately we can, at best, do but little more than alleviate the sufferings of the patient; for when once a collection of matter has formed in the liver, the pathological conditions upon which it depends are, as a general rule, beyond the physician's control. However, notwithstanding that a cure, or even a recovery, may in the majority of cases be unattainable, we must in no case fold our hands in complacent idleness, but be 'up and doing.' For

even in the very worst of cases, as long as the thread of life remains unbroken there is always hope, and we always have it within our power not only to soothe the patient's passage to the tomb, but very considerably delay the fatal issue.

In a wide subject like that of hepatic abscess, which embraces so many and so varied phases, it is impossible to give a succinct *résumé* of all the different plans of treatment. So all I shall attempt to do is briefly to portray the lines along which I myself usually travel. The fact of course of my following these lines being the best proof I can offer of my possessing confidence in them; and it is at least a cheering thing to be able to say that a disease which, when fully developed, is fatal, can, in the majority of cases, be completely aborted if diagnosed correctly and treated energetically in its incipient stages.

The chief lines of procedure I venture to recommend in this formidable form of hepatic disease are :—

1st. If called to the case early—that is to say, when suppuration is threatened, but before pus has actually formed—try to arrest the onward progress of the disease by leeching, cupping, and the application of a freezing mixture of pounded ice and salt over the most pronounced seat of pain. Not only is the freezing process to be continued until the subjacent parts are frozen quite hard, but until actual blistering

of the skin subsequently takes place. As then, and then only, is the cold communicated to the deep tissues of the liver sufficiently intense to abort in the majority of instances the suppurating process.

2nd. In this, the incipient stage of the disease, avoid the application of hot fomentations and poultices, as they only favour instead of retarding the formation of pus.

3rd. Administer a brisk mercurial purgative. Enjoin strict rest of body and mind. Put the patient on low diet, and keep the room well ventilated and of a temperature of not more than 60° Fahr.

4th. Prescribe germicides in the form of salicylic, carbolic, or mineral acids, and quinine, and carefully eschew the administration of alkalies, in any form whatever, as they favour instead of preventing germ development, as well as suppuration.

5th. If the case be not seen until matter has actually formed, the chances of cure are but small, for we as yet know no therapeutical agent which has the power of inducing pus absorption. All we can do is to try the effects of the application of iodine liniment, mustard poultices, or blistering, in the, I fear futile, hope not only of arresting the further progress of suppuration, but of favouring the reabsorption of the already effused pus.

6th. Artificial evacuation is, I believe, the only way of getting pus out of a human liver.

7th. Believing, as I do, that evacuation of the contents of the abscess is the only possible way of getting rid of the pus, I shall go into this mode of treatment fully.

In the first place I may as well observe that to evacuate the contents of an hepatic abscess is not always an easy matter, from the simple fact that, even after the existence of pus has been correctly diagnosed, its exact seat may still remain a mystery. Luckily for us, we not only possess a knowledge of the use of the exploring needle, but are at the same time in possession of the invaluable fact that an exploring needle may be thrust, not alone once, twice, or thrice, but even half a dozen times, within as many minutes, deep into the substance of the human liver with perfect impunity. Dr. J. C. Cameron has the merit of having pointed out (in the 'Lancet' of June 6 and 13, and of August 8, 1863) that 'the liver may be punctured deeply with an ordinary trocar without any evil consequences; the greatest inconvenience witnessed after many such operations being slight local irritation, requiring a few leeches over the wound—and that but very rarely.' He adds that 'in cases where the abscess sought had been missed, and the patient died subsequently of hectic and exhaustion, it was extremely difficult to trace the marks of the trocar in the liver, there being no signs of peritoneal irritation or effusion of any kind.' From this

it is seen that when the abscess is not encountered in the first exploratory attempt, the repetition of it half a dozen times is perfectly safe, unless under exceptionally unfavourable circumstances.

When the case appears to be a very doubtful one, and there is a strong suspicion that more than two exploratory punctures may be required, it is well to use an anæsthetic, and the safest that has as yet been devised is the one recommended by the Chloroform Committee of the Royal Medical and Chirurgical Society (at my suggestion). It is composed of alcohol one part, chloroform two parts, and ether three parts. A formula easily impressed upon the mind by making the quantities of its ingredients rhyme with their initial letters. Thus the initials of alcohol, chloroform, and ether, rhyme as

A C E = 1 2 3

And be it remembered, this is not only a perfectly safe, but at the same time an effective anæsthetic to administer on a pocket-handkerchief. The next point is the selection of the spot for the first puncture. This ought to be the highest part of the bulging. No matter whether it be in the epigastric, lateral, or dorsal region. When there is a bulging in any of these situations, no difficulty can be felt about the matter. Sometimes when the abscess is near to the edge of the right lobe, merely an indistinct oblitera-

tion of the intercostal spaces is discernible. Fortunately, however, this is the spot where least risk is encountered. So that a long fine trocar may be thrust in at this point fearlessly. When the indistinct bulging, on the other hand, is in front, and in the neighbourhood of the gall-bladder, one naturally feels a little more anxious as to the result, in case the gall-bladder instead of an abscess should be punctured. But even little risk attends that accident; for, as will be seen in the chapter on gall-bladder diseases, an escape of bile into the abdominal cavity gives rise to no serious consequences whatever. (Page 1101.)

The following successful case of evacuating an abscess of the liver is recorded by Mr. Arthur Wear :—

In August 1879, a milliner, aged 45, complained of feeling ill. On September 20, the temperature was 102·4°, and pulse 108. On the 21st, she had a rigor, and complained of a sense of great constriction around the diaphragm, which caused severe dyspnœa. A tumour was bulging out, and fluctuation could be distinctly felt through the abdominal wall. On the 22nd, Mr. Wear and Dr. Wicks, using the hand-spray, made a preliminary incision through the skin with a bistoury, and plunged a trocar and canula into the most prominent part of the swelling. About thirty ounces of pus mixed with bile passed through the canula, and the patient expressed herself as greatly

relieved. The temperature fell to 99·2° by the evening, and her pulse to 84 ; she was almost free from pain, but still the tumour did not entirely disappear. On the 23rd, the temperature rose, and, on the 25th, stood at 103·4°. She had a rigor. On the 26th, by means of Potain's aspirator, six ounces of pus and bile, which had reaccumulated, were removed. For many days the temperature remained about 102°, and, on October 5, it rose to 103·4° ; but it gradually fell. By the end of November she was mending rapidly, all traces of the swelling having entirely disappeared.

It must not be supposed, however, that all cases are as successful as this. Sometimes death follows the operation, even when it is skilfully performed. This is usually owing to the operation having been deferred until the patient has become weak and cachectic.

In exhausted, weak, and cachectic patients, the aspirator ought to be used instead of a free trocar, from its being less likely to excite the trifling amount of constitutional disturbance which sometimes follows the evacuation of purulent matter from an hepatic suppuration in badly constitutioned individuals. Some go so far, in employing the exploring needle, as even to attach it to the aspirator.

In no case would I recommend the adoption of the proposal of Bégin and Récamier to attempt open-

ing an hepatic abscess with a scalpel; nor do I advise the use of a large trocar. Not even when the abscess has pointed. For the same advantages may be gained with a small trocar (just of sufficient diameter to allow pus flocculi to escape), and that too with less danger and inconvenience.

As I am writing for the sake of poor patients quite as much as for the advantage of my medical brethren, I may here briefly cite a case illustrative of the danger of opening an abscess of the liver by means of a scalpel, even as a mere auxiliary to the aspirator.

The case I refer to is one recorded by Dr. Neil Macleod, of Shanghai, in the 'British Medical Journal' of November 27, 1880, under the title of 'Hepatic Abscess opened antiseptically.'

A man aged 39, who had been a free liver, had taken little or no exercise for some years. In July 1879, had a severe attack of diarrhœa; in the end of August, an acute attack of dysentery. In November, temperature varied from 97·8° to 103·8°. The pulse varied from 80 to 104. He had night-sweats. There was no rigor or shivering at any time. On November 11, abscess of the liver was suspected, from the increased liver-dulness, fever, sweats, tenderness and pain on the right side. Slight bulging was seen over the lower ribs in the mid-axillary line and behind it; and in this region there were flattening of the intercostal spaces, and a tender spot.

November 14, Dr. Macleod, with antiseptic precautions, passed the largest-sized Matthieu's aspirator-needle between the seventh and eighth ribs in the medio-axillary line, at the tender spot. At a depth of little more than an inch, yellow pus flowed through the tube. Aspiration was immediately stopped ; and, the canula being left *in situ* as a guide, he made an incision on each side of it, and cut down until pus began to well up by the side of the canula. Having removed the latter and enlarged the opening with a probe-pointed bistoury, fully a pint of thick yellow odourless pus flowed from it, with the aid of pressure on the epigastrium. A drainage-tube, with a calibre of half an inch, was introduced. From December 1 to December 20, he had two severe rigors, with an interval of a week between, each followed by a change from liver-coloured discharge to a fresh yellow pus, lasting for two or three days, and then becoming once more dark-coloured, remaining aseptic throughout. Strength began to fail, and the appetite was lost ; and, finally, severe diarrhœa set in. Death occurred on the 20th. *Post mortem*, the back part of the right lobe, opposite the wound, was occupied by a cavity nearly twice the size of a hen's egg, having prolongations backwards and inwards ; one nearly two inches long, admitting the finger.

I may now mention that after pus has been withdrawn from an hepatic abscess (when no drainage-tube

has been considered necessary), a hypnotic dose of bromide of ammonium or chloral ought to be administered to the patient, and he ought to be kept quiet, and be told to lie on the side of the puncture, as pus often discharges itself by driblets through the wound for days afterwards.

The dangers attached to hepatic abscess do not always terminate even when they have been successfully opened. Flint[1] alludes to a case where, after an abscess pointed externally and was opened, it subsequently perforated into the stomach, and the patient died from inanition.

A slow recovery is always to be looked for, even after a most successful evacuation of matter from the liver. Four, eight, or even twelve weeks often elapsing before the patient can leave his bed and move about his room. This arises from the fact that the functions of the liver are in these cases always slow in resuming their normal course.

It being merely the traumatic and, strictly speaking, idiopathic forms of hepatic abscess that can be regarded in the light of local affections (assuredly not those generally spoken of as metastatic or pyæmic, which are undoubtedly due to the impregnation of the constitution with purulent poison), it is the artificial evacuation of them alone that can be expected to be attended with salutary results. And I think

[1] *Practice of Medicine*, 4th ed. p. 551.

in all suitable cases operative procedure ought early to be had recourse to. For, even as a mere palliative, when the evacuation is made early, its benefits are by no means to be despised. By the term 'early,' I mean before the constitution of the patient is broken down by the exhausting effects of the suppuration.

The reason why the artificial evacuation of pyæmic abscesses is attended with no benefit springs from the fact of the constitutional purulent impregnation not being done away with by the withdrawal of matter from one of its mere local foci.

When to attempt the artificial evacuation of an abscess of the liver is deemed unadvisable, either on account of its true seat being unrecognisable, or on account of its nature or the condition of the patient being unfavourable, all our efforts should be directed to induce it to point and discharge itself, either externally, or, what is even quite as good, into the digestive canal. To materially influence its course is unfortunately impossible, but to encourage and hasten it along its own elected route is abundantly within our power. For just as the maturation and pointing of abscesses in other parts of the body may be materially assisted by artificial means, so in like manner can the maturation and pointing of an hepatic abscess be either hastened by judicious, or retarded by injudicious, means. Among the former

may be reckoned the continuous application of heat and moisture. Hot linseed poultices, hot water fomentations, hot turpentine stupes, all of them being applied as hot as ever the patient can tolerate them, and directly over the seat of pain, are the most successful local means of coaxing an abscess to point and burst. But as to do so takes time, and every day the suppuration lasts the patient's strength diminishes, and with it his chances of recovery fade, we must associate the local with an energetic constitutional line of treatment. Which is to soothe and strengthen him, by giving him amusement without excitement, and nutrition without stimulation.

The ventilation and temperature of the room in the early stage of suppuration I have already spoken of. Now I have to point out that a slightly higher temperature is requisite when the object in view is to favour the progress of an abscess of the liver towards bursting. Then it is that a room temperature of from 65° to 70° Fahr. is not too much. But the true index to temperature are the feelings of the patient.

Sleep must be encouraged by hypnotics, chloral. bromide of ammonium, and such like. Not, however, by opiates. The diet should be milk and eggs, animal and vegetable soups, and no solids, except white fish, such as whiting, haddock, sole, turbot, cod, &c. No mackerel, eels, or salmon, lobsters or crabs. Nothing

indeed that will in the least degree tax the digestive powers.

As regards drinks, no stimulants whatever in the shape of brandies or whiskies, unless their employment be distinctly indicated. No port wines or so-called dry sherries. No heavy indigestible malt liquors. But in their place light nutritious feebly alcoholic drinks. Good sound claret, hock, tarragona, or moselle. Effervescing drinks in moderation. That is to say in small quantities at a time—a small wine-glassful may be given frequently. Of sparkling drinks there are a great variety, and they may be selected according to the palate of the patient, so long as none of them are of the acid character sold under the name of *très-sec* and *brut* champagne, the true nature of which I gave my ideas of in the chapter specially devoted to dietetics. Sparkling Devonshire cider (not sweet) included. I prefer really good (not sour) French champagnes to sparkling hocks, moselles, burgundies, &c. &c. With this general exposition of the principles of treatment, I conclude my somewhat long chapter on abscess of the liver, and pass on to one of almost greater importance and equally interesting.

CHAPTER XVIII.

CANCER OF THE LIVER.

No single word in medical nosology conveys to a patient such a chill of horror as that of CANCER, when it is attached to the diagnosis of his case. Consequently it is a word which a medical practitioner ought never to let fall from his lips in a sick room in connection with a liver case, except under very exceptional circumstances, and not even then unless he has indisputable data to rely upon. For, as will be presently seen, the diagnosis of hepatic cancer, even in experienced hands, is often a very difficult task, from the recognition of its existence depending nearly as much on the appreciation of negative, as on the recognition of positive, signs and symptoms. I therefore advise my younger brethren not only never to give a decided opinion that any given case is one of cancer of the liver without having the strongest reasons for so doing, but, even when they have, to break the subject to the patient's friends rather than to the patient himself.

I am led to be thus emphatic from knowing not alone that there is scarcely a single form of hepatic disease—I might even say of any disease—which is so puzzling for a young practitioner to diagnose correctly as a case of malignant disease of the liver, but that of all forms of affection that human flesh is heir to, there is not one about which if he should unfortunately make a mistake, his professional reputation is more likely to suffer. This arises from the fact that almost every old woman imagines that 'a cancer' is a disease easily recognised, and that therefore it ought to be about as plain to the eye of a doctor as a dropped stitch in knitting would be to hers. Consequently, should the poor doctor err, he is almost certain to have his blunder most uncharitably criticised. Here then is a dilemma ; for while on the one hand I tell him not to communicate his suspicions of the case being one of cancer, I nevertheless warn him that if he fails to communicate his knowledge of the existence of that particular form of disease, his reputation is likely to suffer. Fortunately there is an easy way out of the difficulty. Be silent until sure. No sooner feel sure than gently moot your suspicions, not to the patient, but to the patient's friends. Then all will be well.

The sequel will, I think, readily explain why, in spite of cancer of the liver being by no means a rare disease, its diagnosis baffles one more than the dia-

gnosis of almost any other form of hepatic affection whatever. For in studying what is now about to be said on the subject, it will become painfully apparent that while most of the positive diagnostic signs testifying to its presence are common to other forms of liver disease, almost all the negative symptoms in general relied upon as being indicative of its absence may with equal propriety be negatively applied to a host of other hepatic affections. Added to which the signs and symptoms of cancer of the liver hitherto given in text books (as will be seen on comparing them with those presently to be put forward) are not only in the majority of cases defective, but in several instances actually erroneous. Consequently they are more or less misleading guides to the diagnosis of any beyond the plainest of cases. Such being my belief, I will enter more fully than I should otherwise do both into the pathology and etiology of hepatic cancer, in the hope that a knowledge of these branches of the subject will facilitate both the diagnosis and treatment of the disease. While, further, I shall tabulate a few hints which I trust may be useful to the beginner in the differentiation of obscure cases, which I know by personal experience will occasionally tax his energies as well as his abilities to the utmost.

Pathology of Hepatic Cancer.

It is in general asserted, not only in text-books but in special treatises on diseases of the liver, that the hepatic organ is liable to be attacked with the following six forms of cancer only: encephaloid, melanoma, fungus hæmatodes, epithelioma, sarcoma, and scirrhus. On going carefully through the recent British and foreign literature of liver diseases, I have found, however, that every known form of so-called cancerous diseases attacks the human liver. Even the rarest of all the specially named forms of cancer, namely, the colloid variety, which has again and again been said never to attack the human liver, I have met with in at least three reliable well-recorded cases. One is that of a preparation in Guy's Hospital Museum. Another is a case which Dr. Vanderbyl exhibited to the Pathological Society in 1858. Which specimen greatly impressed me, as it was taken from a woman aged 64, who, notwithstanding that her liver was extremely diseased, had shown neither symptoms of jaundice nor anasarca. And yet the transverse fissure was completely filled with colloid growths, while the external surface of the liver as well as of the gall-bladder was studded over with colloid tumours, varying from half an inch to an inch in diameter.

As I shall afterwards attempt to prove that the

irritative effects of gall-stones are among the exciting causes of cancer of the liver, I may incidentally add that forty small gall-stones were found in this patient's gall-bladder, which led Dr. Vanderbyl to the not improbable supposition that the irritation caused by their presence in a measure accounted for the peritoneal covering of the gall-bladder having become so prominently affected with the morbid deposits. In this case, however, the presence of the stones could scarcely be suspected of having induced the cancerous disease, as the deposits, both in the gall-bladder and in the liver itself, appeared to be secondary to other similar growths existing in the omentum and ovaries.

The third case is also one in which the hepatic colloid appeared to have extended as a secondary formation from the intestinal tract. The case is reported by Mr. George Lawson in the thirteenth volume of the Clinical Society's 'Transactions.' The liver is described as having been 'enormously enlarged, especially the right lobe, and almost wholly converted into a mass of colloid, which projected from its surface in the form of large irregular tumours. On section, but little hepatic tissue remained.' Yet, notwithstanding this, and the fact that the diseased growths were secondary to a similar morbid degeneration in the sigmoid flexure, and that even the pelvic glands were affected, the left lobe

of the liver, in spite of being enlarged, was free from disease. (The immunity of the left lobe of the liver, not alone from cancer, but from abscess, as well as from hydatids, is a pathological phenomenon to me quite inexplicable. No doubt an anatomical or physiological cause for its immunity must exist, but what it actually is I know not.)

The patient in this case was only 23 years of age, and had had gastro-enterotomy performed on him nine months previous to his death for an intestinal obstruction (supposed to have been caused by a twisting of the bowel) in the neighbourhood of the cæcum. The cancer probably owed its origin to the irritative effects of the previous bowel mischief.

While asserting that the human liver is liable to be attacked with every known variety of cancerous disease, I cannot refrain from entering a protest against the slipshod manner in which tumours of the liver and its appendages are often described as cancerous when not a trace of malignancy exists about them. Everyone conversant with the modern literature of liver diseases will, I am sure, bear me out in the opinion that the common generic title of 'cancer' is oftentimes most erroneously and misleadingly employed in the morbid anatomical descriptions given of liver cases. Every tumour, tissue-thickening, growth, or degeneration of the

liver whose histology is not self-evident, is dubbed a 'cancer.' Again and again have medical men sent to the physiological laboratory at University College specimens of what they were pleased to denominate 'cancer,' which on investigation proved to be nothing more than inflammatory thickenings of the capsule, neck of the gall-bladder, or of the ducts, following upon inflammatory ulcerative action caused by the presence or passage of gall-stones. In fact, not only general practitioners, but men with some pretence to a special pathological knowledge, apply the name of 'cancer' to all species of doubtful growths. Even those which produce no cancerous cachexia or infiltration of the neighbouring tissues, and consequently are not constitutional, but merely local diseases.

There is, perhaps, some excuse for many of these mistakes, from the fact that at the present moment there are no forms of hepatic structural tissue change about which there exist such confused notions as those thought to merit the name of 'cancer.' Everyone knows, for example, that the generic title of 'cancer' is given not only to a wide, but also to an absolutely incongruous, variety of new tissue-growths and degenerations, some of which possess not a vestige of similarity to one another, either visually, microscopically, chemically, or clinically. Some being of slow development, solid in structure,

as hard as a cricket-ball, and giving rise to no constitutional signs. Others are of rapid growth, soft, juicy, and inducing a well-marked constitutional cachexia. How, then, let me ask, can the generic title of 'cancer' be legitimately applied in a clinical any more than a pathological sense to such widely differing morbid structures?

Another and most fruitful cause of error in the nomenclature of hepatic tumours and tissue degenerations in the non-pathologically trained mind, originates in the fallacious belief that all forms of new growth or tissue degeneration, which inevitably lead to a fatal termination, 'must be a cancer.' After having said this, I think it incumbent on me to try and answer the question—

What is a Cancer of the Liver?

It may perhaps be as well for me to take the reader at once into confidence, and show him the true colours under which I sail. They are these:— It is my opinion that the name of 'cancer,' not only as regards the liver, but equally as regards every other organ of the body, ought only to be given to those forms of new tissue formation which are clearly of a constitutional in contradistinction to a merely local origin, as testified by their tendency to affect the glands, infiltrate the neighbouring tissues, and produce the so-called cancerous cachexia. Thus

a cancerous tumour of the liver, to my way of thinking, is an abnormal constitutional growth, totally distinct and entirely different in its pathological characters, as well as in its clinical signification, from a merely, though, it may be, necessarily, fatal form of liver tumour. For plenty of perfectly benign forms of growth affecting the human liver are, on account either of their size or position, of necessity fatal. None of these, however, give rise to what is called a cancerous cachexia, and to the form of tumour alone which possesses this power would I, under any circumstances whatever, give the title of 'cancer.'

As I entertain very decided views on this subject, and as I believe that cancer—that is to say, true malignant disease of the liver—differs in no single particular from malignant disease in any other organ of the body, except in so far as the histological elements of the normal tissues it attacks slightly modify its naked-eye and microscopical characters, I shall here reiterate some of the views I have so freely expressed in the morbid anatomy part of my 'Histological Demonstrations,'[1] specially devoted to the consideration of the microscopical characters of various morbid growths; for I believe the universal adoption of the doctrines there propounded would not only advance the progress of pathological ana-

[1] Second edition, p. 180. Longman & Co., London.

tomy, but greatly facilitate the correct interpretation of many at present seemingly obscure clinical data in connection with a variety of so-called cancerous diseases. Moreover, I believe that it will not only tend greatly to assist the reader to follow the lines of argument I shall pursue in giving the etiology of hepatic cancer, but also enable him all the more readily to understand the clinical histories of the cases I shall cite, and the therapeutical bearings of my subsequent remarks, if he attends to the present exposition of my views on the nature of hepatic cancer. So he need not regard their perusal as an unnecessary waste of time. They are :—

1. In diseased livers neither are new histological elements created, nor are new functions developed ; but existing tissues are only modified or misplaced, and their normally existing functions disordered.

2. All liver, like other tumours, grow in the direction of the least resistance. Where there is entire freedom from pressure, the form of the tumour is globular, as is the case when it grows outwards from the liver into the peritoneal cavity.

3. All liver tumours are liable to alteration of texture in the course of their growth. A fibrous tumour may become softened, cystic, cancerous, or calcified, a hard and benign scirrhus may either become fattily degenerated, or gradually transformed into a soft true encephaloid cancer.

I have on several occasions, while examining cases of malignant disease of the liver, been struck with the very marked tendency encephaloid tumours have to become softened in their centres into a white opaque creamy liquid, making the tumour assume an almost cystic character. Sometimes the interior of the cyst appears to be divided into sections by fibrous partitions, giving to the tumour a multilocular appearance. When several growths existed in the same liver, they would, according to their degrees of advancement in the degenerating process, show the different stages of the gradually softening disorganisation going on in their interiors.

4. Benign liver tumours show no tendency to infiltrate the neighbouring tissues or to affect glands.

5. In true scirrhus, which is a benign form of growth, there is no affection of the glands, no infiltration of the tissues, and no 'cancerous cachexia.'

6. All kinds of morbid growths of the liver have a tendency to run into each other. The characteristic features of each becoming gradually less and less distinct, until ultimately the benign appear to be almost identical in character with the malignant.

7. Malignant liver tumours affect glands, infiltrate the neighbouring tissues, and induce a 'cancerous cachexia.'

8. The accession of malignancy in the course of

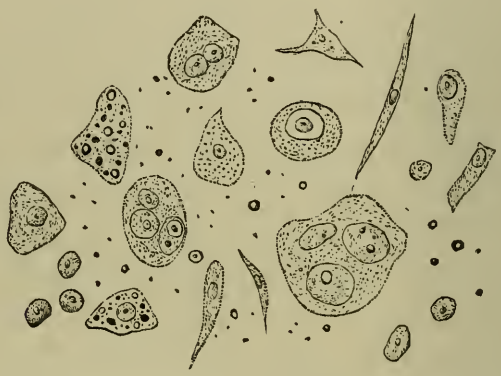
any case of hepatic disease can be recognised by the supervention of the 'cancerous cachexia.'

9. When examined after death, there ought to be no difficulty (although I regret to say there very often is) experienced in recognising hepatic cancer, from the simple fact that all truly malignant forms of growth present the following well-marked naked-eye and microscopical characteristics.

First as regards the microscopic elements. At one time there was a universal implicit belief in the existence of an essentially characteristic form of cancer-cell, the presence of which in any tissue or fluid of the body was thought to be indubitable evidence of the existence of malignant disease. Then came a time when this supposed fiction was totally abandoned. The advancing wheel of pathological knowledge has, however, again revolved. A fresh epicycle in morbid histological thought has been reached, and I am now, as I have no doubt many others are, fully prepared to affirm that the existence of a veritable specific form of cancer-cell is not a conceit of the imagination, but it is as easily and infallibly recognisable by the eye of the initiated as the different individual members of a flock of sheep are to the eye of their shepherd, though at the same moment they may be totally undistinguishable to the eye of an outsider. The subjoined figure, copied from the morbid anatomy part of my 'Histological Demonstra-

tions,'[1] shows exactly the kinds of cells upon which implicit reliance may be placed in diagnosing a malignant tumour. There are many benign cells bearing a resemblance to those here depicted ; but there is not a single cell to be met with, either in the diseased or in the healthy human body, identical with these, except

FIG. 25.



Encephaloid Cancer-cells. With Brownian Granules.

in connection with cancer—that is to say, as I define it, a true cachexial constitutional form of disease.

Mark these words ; for they must not be misinterpreted. They have no reference whatever to any of the other, erroneously, so-called 'cancer cells.' They have nothing to do with the constantly misinterpreted pathological elements met with in growths called scirrhus. Which, in my opinion, has no more right to be designated 'a cancer' than either an adenoma or a fatty tumour has. This idea can be

[1] 2nd ed. p. 224.

proved to be a legitimate one; alike experimentally, histologically, and clinically. Thus, for example, inoculated encephaloid cells or juice beget cancer. While inoculated scirrhus cells or scirrhus exudation (there is no scirrhus *juice* proper) beget—NOTHING.

Langenbeck and Lebert found cancerous tumours form in animals inoculated with cancer cells and cancer juice. A fact not in the least difficult to understand, for recent cases have been reported as having occurred which illustrate in a remarkable degree how cancer germs may be transmitted even, in some as yet unknown way, from one organ to another not only having no direct connection with each other, but possessing no similarity either in their structure or functions. Secondary cancerous deposit affecting the entire pulmonary tissue having been traced to primary encephaloid in the spleen. In the same way encephaloid growths upon the lip and in the mammary gland develop the disease in distant lymphatic glands.

The mere contact of a cancerous growth of one organ with the surface of another is, in some instances, sufficient to propagate the disease. For we sometimes meet with encephaloid of the liver inducing encephaloid in that portion of the mesentery with which the organ is in intimate contact. Thereby showing that when the constitution is saturated with the cancerous cachexia, the transudation of morbid fluids into a healthy structure in mere contact with a diseased

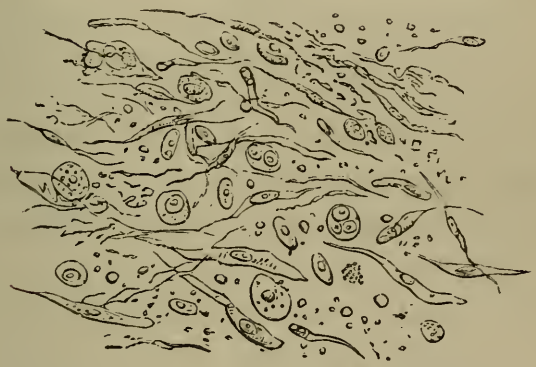
one is sufficient of itself to induce the development of malignant growths. Numerous other instances of encephaloid cancer spreading by mere contact (from the stomach to the liver, and *vice versâ*, for example) could be adduced. But who can point to a single case where a scirrhous tumour has ever propagated its species either by contact or any other means whatever?

When Mr. Z. Lawrence was preparing for the press his book on cancer, I performed a number of experiments for him on the inoculability of cancer, among which was the following crucial one with scirrhus. The result of which completely negatives the idea of scirrhus being a malignant disease.

The experiment was planned with the object of testing the possibility of communicating scirrhous disease from one living mammary gland to another by keeping a freshly made surface, two inches in diameter, of a living scirrhous tumour in close contact with an equally freshly made surface of a healthy living mammary gland of an animal of the same species. Although this was done for thirty minutes, and at the end of that time the two mammary glands were found to be in a measure glued together by the exudation from their cut surfaces, not a vestige of the disease was communicated from the scirrhous tumour of the diseased to the healthy mamma of the other dog. At least during the year the animal was kept under observation. I may further mention that I

have repeatedly injected scrapings from scirrhous tumours freshly removed from the human subject into the veins, as well as under the skin, of healthy dogs, without on one single occasion having so much as seen more than a trivial temporary inconvenience arise from the inoculation. Most assuredly not so much as a vestige of scirrhus or any other form of tumour was ever discoverable in the bodies of

FIG. 26.



Elements of Scirrhus from a Human Mammary Gland.

any of the animals experimented upon. Which were killed, and carefully examined at varying periods from one to eight months after the inoculation had been made.

As regards the histological elements of scirrhus, again, they bear not the remotest resemblance in any respect whatever to those of true cancer, as before depicted (fig. 25). For they are spindle-shaped fibre cells, with no large nuclei or Brownian granules. The

accompanying fig. 26, which is also from the morbid histological part of my 'Demonstrations,' shows roughly, though exceedingly well, the elements met with in an ordinary scirrhous tumour.

In deciding upon what tumours of the liver ought, and upon what ought not, to be considered malignant, it may be convenient to recollect the following characters, which, when coexistent, at once decide the growth to be malignant in its nature.

a. The tumour has a milky juice, which, under the microscope, is found to be full of variously shaped large granular nucleated cells (fig. 25).

b. The growth has no well-defined outline, but gradually shades away into the surrounding tissues.

c. It is generally soft and pulpy. Mottled red and white.

d. The neighbouring lymphatic glands are certain to be affected if the disease has existed for any length of time.

e. The disease is associated with the 'cancerous cachexia.'

Finally,—The disease to which I opine ought to be restricted the name of cancer is a constitutional and not a merely local affection. Its local manifestation in any one part or in any series of parts of the body being exactly analogous to the local manifestations of small-pox, glanders, contagious jaundice, or any other form of germ diseases, of which I regard

cancer as being one. Consequently, as the vice is in the patient's system, the removal of the merely local manifestation of a true cancer by the surgeon's knife or by the empiric's caustic is an uncalled-for piece of cruelty. Just as much as would be the excision of a malignant pustule. The operation, instead of retarding, invariably (in consequence of its weakening effects on the patient's constitution) hastens, the fatal end. For no matter however skilfully the local manifestation may be extracted, its removal can never eradicate the vice from the system. Hence it is that a local manifestation of true cancer always recurs either in the same or in some other part of the patient's body; not once merely, but again and again after each successive operation for its artificial removal. Which fact of itself is proof positive that cancer is a constitutional and not a merely local disease, as some would have us imagine.

While studying the subject of cancer of the liver, it is essential to remember that strange forms of tissue metamorphosis may occur in the organ. For example, a simple inflammatory tissue degeneration may change into a hard scirrhous tumour. Which in its turn may gradually become metamorphosed into a soft juicy encephaloid. There are but few exceptions to this general law of developmental change. But fortunately one does exist, and that is, that encephaloid parasitic germs sometimes die, and, like the

larger forms of parasites (trichina, hydatids, &c.), spontaneously undergo calcareous cretification.

As the history of a nation repeats itself, so do the doctrines of its people, and it will be noticed that my saying a cancer is a germ disease is tantamount to saying that it is a parasitical form of disease. And in so saying I but resuscitate the old doctrine that a cancer is an animal ! I think that I showed in my series of papers on germs, in the 'Lancet' (June and July 1881), that Brownian granules (the form of germs so constantly present in all varieties of encephaloid and melanotic cancers) have a much stronger claim to the title of animal than of vegetable germs.

Hospital dead-house statistics show that the liver comes next to the uterus as regards its proneness to be affected with those forms of tissue degeneration which have hitherto been grouped together under the common name of cancer.

Etiology of Hepatic Cancer.

This is a most important subject both in a diagnostic and therapeutical point of view, for a knowledge of the exciting causes, as will presently be seen, materially facilitates not only the diagnosis, but even the treatment of obscure cases.

The most common of all exciting causes of hepatic cancer is hereditary predisposition. And, in order to raise the suspicion of the existence of this important

diagnostic factor, it is not at all necessary to be able to discover that a parent or grandparent, a sister or a brother, an uncle or an aunt, have suffered from the same form of disease. All that is requisite to furnish legitimate grounds for the suspicion is that some blood relation, no matter how distant, cousin or half cousin, has been affected with some form or another of malignant disease in any part of the body—the brain or the stomach, the uterus or the ovaries, the testicles or the mammæ, or even in the limbs. For it matters not one whit where the cancer has been located. The mere fact of its having existed being of itself sufficient to raise the suspicion of hereditary taint. For the locality in which the visible manifestation of the constitutional disease appears is in many instances the result of mere accident. For example, a blow on the breast may induce a cancer to form in that locality in one, while a blow on the liver will induce an hepatic cancer in another member of the same family.

Moreover, it is well to bear in mind that a cancerous inheritance may descend through several generations, even though missing more than one in direct succession.

It is a noteworthy fact, too, that cancer appears to be common in tuberculous families. Frequently tuberculous parents give birth to cancerous children, and in connection with this point, after what was

said regarding the prevalence of Brownian granules in encephaloid and melanotic cancers, I may mention that not only I, but several other microscopists, have noticed how exceedingly common not only Brownian granules, but the next stage of their higher development—namely vibrios—are in the sputa of advanced phthisis.

In persons constitutionally predisposed to cancer, a mere trifle seems in some instances to be sufficient to call it into existence in the liver. Not once, but on several occasions, patients have attributed the presence of the cancerous growth in their liver to an antecedent injury to the organ. Indeed, the more attention I give to this point, the more impressed do I become with the belief that mere mechanical injury to, or irritation of, either the nerves or tissues of the liver is a far more frequent exciting cause of its malignant degeneration than the majority of physicians imagine.

I suppose that all or nearly all surgeons are now agreed that direct injury to female mammæ is a common cause of cancer of the breast. So I shall now do my best to make physicians equally alive to what I believe to be a fact, namely, that injury to, or irritation of, the liver is often followed by malignant disease in that organ. I shall cite cases in support of this opinion. And, to begin with, I quote what I regard as a typical example. It is recorded by

Dr. Pye Smith in the thirty-first volume of the Pathological Society's 'Transactions.'

It is there stated that a healthy country boy, aged about 12 years, three months after having had a fall on his side noticed a swelling, which gradually increased and gave pain. It was accompanied with fever and occasional coffee-coloured urine. He died in fifteen months after he first sought advice; that is to say, about sixteen or seventeen months after receipt of the injury. At the autopsy, although the boy was only twelve years of age, his liver weighed two hundred ounces. The cancer consisted of 'circumscribed masses of soft yellow material,' and was limited to the right lobe.

Having already referred to a case where cancer of the liver followed upon the irritation caused in the digestive canal by the performance of gastro-enterotomy, and to another where it was associated with the presence of gall-stones, I will ask the reader to peruse the chapter on cancer of the gall-bladder, as he will there find other illustrative cases, among which is an interesting one where the presence of gall-stones appears to have been the direct exciting cause of cancer both in the liver and in its bile-ducts. While, from the history of the following case, it even appears as if a mere hepatitis were sufficient to excite the development of cancer of the liver in a predisposed constitution. For among four cases of primary cancer

of the liver, occurring in natives of India, related by Dr. Ewart in the 'British Medical Journal' of September 18, 1880, there is one strongly suggestive of this opinion. It is as follows :—

A Hindoo woman, aged 45, whilst in the enjoyment of good health, was seized with hepatitis, and suddenly became jaundiced, with porter-coloured urine, white evacuations, and itchiness of the skin. Jaundice was so extremely developed that a yellow tinging was observed in the tears, as well as in the mucous lining of the mouth, fauces, and in the nails of the fingers and toes. There was yellow vision. The liver reached to the iliac crest below, and to the nipple above. There was no pain or tenderness. Purpuric spots appeared on the face, hands, arms, and trunk; the kidneys gradually struck work; coma came on; and, after remaining in a state of insensibility for two days, she died.

On *post-mortem* examination, the liver weighed one hundred and thirty ounces. It was nodulated from cancerous growths. The glands around the common duct were enlarged from cancerous growth, causing the complete obliteration of it and of the cystic duct. The gall-bladder contained a small quantity of pale-coloured fluid. Some of the bile-ducts were enormously dilated, and distended with bile. The left lobe was free from cancerous material. Every other organ was healthy.

In connection with the exciting causes of cancer

I may mention that it is my opinion that cancer-germs, like other disease-germs (as I showed in the series of papers I published in the 'Medical Times and Gazette' in November and December 1881, on the action of germs in the production of human diseases), may lie latent or dormant in the system for years, and yet at length be called into sudden developmental activity by some trifling local irritation or constitutional disturbance.

I have now to call attention to another important fact in connection with the etiology of hepatic cancer. Which is, that while the liver may be the seat of soft encephaloid, some other organ of the body may at the same moment be affected with what is called a hard cancer. And what is more curious still is the possibility of the soft cancer owing its origin to the irritative effects of the pre-existing hard growth.

A case of this kind came under the notice of Mr. Nunneley in 1858. The patient, an elderly maiden-lady, had first a painful scirrhus of the right breast, and then in a year later began to complain of acute pain in the region of the liver. She died within the second year, apparently from exhaustion, and at the *post-mortem* the liver was found three times its natural size, and infiltrated throughout with medullary fungoid matter. The latter soft form of disease, though apparently not only supervening on, but depending upon the pre-existence of, the hard tumour,

yet far outstripping it in rapidity of growth. It has long been known that two allied forms of cancer might even exist in the liver at one and the same time ; for cases had again and again been met with where a melanotic degeneration was associated with an encephaloid cancer. This pathological fact, however, is not, and cannot be, regarded as anything extraordinary. Seeing that a melanotic degeneration is often a mere sub-form of pigmentation of an encephaloid tumour. Nunneley's case was, as seen, quite different from this, and is exceedingly valuable in teaching us the important clinical lesson, never to imagine, because we have a hard scirrhous growth in a visible organ, that its secondary form of growth attacking the invisible liver must necessarily possess the selfsame morphological elements and pathological characters.

Signs and Symptoms of Cancer of the Liver.

Before detailing the symptoms usually given in text-books, I wish to call special attention to four essential factors in connection with the diagnosis of malignant disease of the liver which we are not nearly, I consider, sufficiently alive to. Namely :—

1. That true malignant disease of the liver may, and actually does, occur at any period of life between the cradle and the coffin.

In proof of this I may refer to the case recorded

by Dr. Grouse ('Philadelphia Medical and Surgical Reports,' 1874) of a child who was found to have encephaloid disease of the liver when it was less than five months old. The tumour was nodulated, increased rapidly, and gave rise to the cancerous cachexia. The infant died when it was about a year old, and at the necropsy the liver was found softened, and is said to have actually weighed between fifteen and twenty pounds! and to have nearly filled the whole abdominal cavity. A microscopical examination of its tissue proved the disease to be encephaloid. Two aunts of this child had died, it was said, of scirrhus; but both its parents were quite healthy.

Cases of this kind in infants are exceptional. Indeed, it might almost be given as an aphorism that malignant disease of the liver increases in relative frequency in direct proportion as age advances.

2. In the vast majority of cases cancer begins in, and is entirely limited to, the right lobe of the liver.

3. In all cases of malignant disease of the liver, at any rate towards their latter stages, there is present more or less of a well-marked cancerous cachexia.

4. Not alone is jaundice by no means a necessary concomitant of hepatic cancer, but it is in reality seldom associated with it. For by a calculation I have made of reported cases it seems that no less than ninety-four out of every hundred are unaccompanied

by a distinctly icteric tint. In not more than about six per cent., therefore, of cases of hepatic cancer is jaundice encountered. This statement may appear to be startling intelligence to some, but it is nevertheless quite true, as can be shown by reference to hospital dead-house statistics. Thus, for example, those of the Middlesex Hospital, which were published by Dr. Vanderbyl in the ninth volume of the Pathological Society's 'Transactions' (p. 234), show only two cases of jaundice in a total of twenty-nine of hepatic cancer.

The reason of this rarity of jaundice in cases of cancer of liver is readily explained when it is recollected that, as I previously showed, jaundice can only occur from a blocking-up of the hepatic or common bile-ducts, or from a total arrest of secretion; and cancer seldom either completely occludes the ducts or destroys all the bile-secreting structures.

The first symptom, and one upon which great stress is usually laid in text-books, is that of pain in the right hypochondriac region, and no doubt it is a constant one ; but pain is so common an accompaniment of hepatic disease, that it is necessary for me to say something more about it than is generally said in books. To begin with, I may state that it is to be differentiated from the pain produced by gall-stones, for which it is often mistaken, and *vice versâ*, by its being neither acute nor paroxysmal, in general being

described by the patient as of a dull aching character. Firm pressure always increases it, and sometimes causes it to assume an acute and stinging character at the part immediately beneath the seat of pressure. In hepatitis (for which the pain of cancer is apt to be mistaken), the pain is not only more acute, but at the same time it is invariably accompanied by febrile symptoms. The only other form of pain with which that arising from cancer is likely to be confounded is that associated with abscess ; but here again the difficulty in differentiation is but slight, when it is remembered that rigors and hectic are the almost invariable accompaniments of suppuration of the liver.

The next sign of hepatic cancer upon which emphasis is usually put is enlargement of the affected organ, or part of the affected organ. For the whole liver is seldom implicated, from the simple fact that the patient usually succumbs before the disease has spread itself over the whole organ—though there are even exceptions to this rule.

The sign of hepatic enlargement, though of some value in the majority of cases, is unfortunately not to be depended upon, from the fact, as I shall now proceed to show, that even fatal forms of hepatic cancer may be present, and yet the liver, instead of being above, be actually below the normal dimensions.

My former colleague, Professor Charles Hare, had a case where the whole cancerous liver weighed only twenty ounces! And Dr. Fagge has recorded another, which he named primary contracting scirrhus of the liver, weighing thirty-six ounces and a half. It came from a man aged 52 who had ordinary ascites. On *post-mortem* examination was found a granular cirrhotic-looking liver. Some of the lobules contained cheesy-looking matter and were friable. There was a thrombus in the portal vein. (See remarks at p. 1030.)

At the February (1881) meeting of the Birmingham Branch of the British Medical Association, Dr. Mouillot showed a liver in which cirrhosis and scirrhus were both present; and the appearances suggested that the scirrhus had supervened upon an already cirrhosed liver.

In contradistinction to these exceptional cases of small liver I shall now cite the startling one already alluded to as having been recorded by Dr. Gordon, where the liver, instead of weighing from forty-five to fifty ounces, as it ought, if healthy, to have done, weighed no less than 380 ounces. The case is recorded in the 'Dublin Quarterly Journal' of November, 1867. It was one of encephaloid disease in a man aged 50, who never suffered from jaundice, and in whom the organ was supposed to have in a single month attained the enormous weight of twenty-four pounds.

According to Dr. Gordon's measurements, the man's liver increased from about six pounds in weight on January 1, to twenty-four pounds on the 31st—the day he died. That is to say, it increased at the rate of nearly ten ounces daily. For Dr. Gordon says that on January 1, 'as far as could be judged from external measurement, it was about one-fourth of the dimensions it subsequently attained.' At the *post-mortem* 'the enlarged liver seemed to occupy the entire abdominal cavity.' The only symptoms at first noticed were pain and rapid emaciation. But within a few days of his death he became anasarcous. The urine was scanty, with a copious deposit of lithates. Two days previous to the patient's death the abdominal pain became so intense that the patient fell into a state of collapse, from which he never rallied ; and this was found after death to have arisen from a perpendicular rent having taken place in the peritoneal covering of the right lobe, of about three inches in extent, from which a quantity of blood had escaped. The liver had lost all its normal shape, and looked like a great ball thickly studded over with cerebriform tumours, numbers of which were also in the lumbar glands, as well as in the peritoneal and pleural cavities.

This is an exceedingly and exceptionally rapidly growing case of hepatic cancer; but it is by no means the only example with which I am acquainted,

for several cases of very rapidly developing encephaloid of the liver are on record. When I say 'rapidly developing,' I mean, the illness from its beginning to the fatal termination not having exceeded a period of six months. Even some of the cases have been subjects under 25 years of age.

It may be said that, as a rule to which there are but few exceptions, cancerous growths in the liver are insidious in their onset, and slow in their progress—which is fortunate for the practitioner in a diagnostic point of view, as it enables him, in diagnosing cancer of the liver, to eliminate from his calculation the acute congestive forms of hepatic enlargement, all of which, malarial and other, as a general rule, are sudden in their onset and rapid in their progress.

As regards the average amount of enlargement of the cancerous liver, I think it may be said not to be great. For on looking over the weights given of different livers, I find that the whole organ, cancerous mass and all, is in general stated to have ranged between sixty and ninety ounces.

The next valuable diagnostic sign of the existence of hepatic cancer usually given in books is nodulation of the abdominal surface of the liver. I believe that far too much importance is attached to this sign, from the fact that not only is it sometimes absent, even in the encephaloid as well as in the colloid varieties of the disease, but it occasionally happens

that a distinct nodulation is perceptible when no form of cancer is present. As, for example, occurs in certain cases of superficial multiple abscesses and hydatids, which render the differential diagnosis all the more embarrassing, on account of their often being not only of exactly the same size—as big as chestnuts or oranges—but communicating to the hand the same sort of indistinct feeling of fluctuation. Fortunately in these cases we have a differentiating guide in the presence or absence of the cancerous cachexia. For, as far as I have been able to make out, when cancerous nodules are perceptible, the system is always so impregnated with the morbific agent as to give evidence of its presence in the complexion. Here, too, the existence or absence of jaundice is a sign of no avail. For it is as frequently absent in cases of hydatids and suppurative disease as it is in those of cancer.

The sign to which I attach the greatest importance in a suspicious case is the existence even of a very slight cancerous cachexia. For it, I believe, is pathognomonic. So that whenever I detect its presence, especially if it is associated with a cancerous family history, I put down the enlargement of the liver, in absence of other evidence to the contrary, as due to malignant disease.

Besides the above-mentioned physical signs, there are also a variety of constitutional symptoms which

may be regarded as aids to the arriving at a correct diagnosis. For example, in malignant cases there is in general some derangement of the digestive and assimilative functions, furred tongue, torpid bowels, flatulence, and nausea. Or the digestive derangement may assume somewhat of the opposite form, and diarrhœa, and even vomiting, be prominent symptoms, leading to the suspicion that the cancer is seated in the stomach instead of in the liver. A circumstance easily accounted for, from the fact that the liver and stomach are such highly sympathetic organs that cancer of the one is often accompanied with cancer of the other. In fact it is generally believed that hepatic is very often secondary to stomachical cancer. Some say that it is so in at least a third of the cases met with.

In the next place, as may be judged of by what was previously said regarding the influence of age on the relative frequency of malignant disease of the liver, the time of life at which the patient has arrived furnishes a not unimportant factor in the diagnosis. For cancer of the liver is, comparatively speaking, rare before the age of 25, and is most common between the ages of 40 and 60. Even infancy, however, as was before said, does not preclude the possibility of the case being one of soft cancer. In addition to Dr. Grouse's case of encephaloid which began when the child was only five months old, I may allude to the one Dr. West has recorded at page 732 of the sixth edition of

his book 'On the Diseases of Infancy.' The patient was a boy only eight months old when he began to lose flesh, and within a month afterwards his mother noticed solid masses in his abdomen. He became exceedingly sallow, and suffered from diarrhœa, which, accompanied as it was with severe pain, killed him at the age of twelve months. The tumour felt in the abdomen, which was larger on the left than on the right side, turned out to be the almost healthy left lobe pushed out of its place by the enlarged and diseased right lobe, part of which was converted into a soft white brainlike matter, intermingled with which were portions of a firmer, highly vascular, fibro-cellular substance. A few deposits of medullary cancer also existed in the right lung.

As a further aid to diagnosis I may specially mention the fact that whenever cancerous disease of the liver is sufficiently advanced even to make its presence suspected, it usually runs a rapid course. I am not alluding to the hard forms of so-called cancer, which are invariably of slow growth, but only to what I have said I consider deserving of the title of true malignant disease. Namely, soft growths.

Negative Signs and Symptoms of Hepatic Cancer.

a. Jaundice is rare, even a slight icteric tint seldom being seen in more than one out of every seventeen cases of the disease.

b. All signs and symptoms of pyrexia are, in cases

of non-complicated cancer of the liver, totally absent. It may be indeed confidently affirmed that fever is never present unless in connection with some form or another of independent disease, as, for example, when inflammatory action occurs as an accidental complication to the malignant disease.

c. Acute pain is seldom or never present.

d. The superficial veins of the abdomen are seldom enlarged, as it rarely happens that the cancerous growths implicate either the vena cava or the main trunk of the portal vein.

e. Ascites is, for similar reasons, also uncommon, except in the very last stage of the disease, when the general debility of the absorbents becomes manifest in this way.

Some of the difficulties encountered in the diagnosis of hepatic cancer having been already made apparent, both in a case of inspissated bile, and in one of gall-stone, in the respective chapters on these subjects, it only remains for me now to adduce a typical example illustrative of the forms of difficulty which now and again beset the path of its diagnosis, in order that it may serve as a danger-signal. Fortunately I have ready at hand a most remarkable as well as a most instructive case. For its history at one and the same time embodies in itself the most salient points of embarrassment as well as of their solution.

The case I refer to is that of a gentleman who was connected with one of our large metropolitan public institutions, which presented nearly all the difficulties in the way of diagnosis it is possible to imagine. So very anomalous indeed were its signs and symptoms that one of our most accomplished hospital physicians diagnosed it for, and treated it as, a case of malignant disease, when not a trace of cancer, as subsequent events proved, was present in his system. I first saw the patient on March 3, 1869, at which time, in spite of his being strong enough to come by himself to consult me, he had all the appearance of a man tottering on the verge of the grave. The account he gave of himself was that for several months previously his health had gradually failed him. The first prominent symptoms that attracted his attention having been a feeling of discomfort, scarcely amounting to actual pain, in the right hypochondriac region, accompanied with a general feeling of *malaise* and loss of strength, which soon increased to such an extent that he could not exert himself either bodily or mentally. These symptoms were associated with a marked sallowness of the complexion, which rapidly merged into a decided jaundice. It was then noticed that his stools were of a pipeclay colour, and the urine of a very dark tint. As he not only felt but looked very ill, and was likewise losing flesh, his medical attendant took a

gloomy view of his case, and called in Dr. (now Sir) George Burrows, who, after a very careful examination, pronounced the case to be one of malignant disease of the liver, which would inevitably end fatally, and that too, as the disease appeared to be progressing rapidly, at no very distant date. Seeing that this weighty opinion exactly coincided with that of the other medical men he had previously consulted, the patient was advised to give up all further attempts at performing his public duties. A piece of advice which, from its being in accord not only with his own feelings but with the opinion of his family, was at once acted upon, and resigning his appointment he retired to a pleasantly situated country house, there to await, as he said, his summons to eternity. Some weeks afterwards he was led by the earnest solicitations of Mr. E. Jacob, surgeon to the Birkenhead Hospital, to come and consult me. This being the brief history of the case up to the time I first saw him, I shall now relate its subsequent history from personal observation. When he entered my study, he looked like a man, as I before said, on the brink of the grave. He was thin, haggard, and sallow. His liver was enlarged, measuring in the perpendicular nipple line over six inches. It was tender, but not acutely painful on pressure, except at one point, and that was directly over the gall-bladder. He complained of all the before-mentioned symptoms

in a more or less marked manner. He had no pruritus as in gall-stone, and no cancerous cachexia as in malignant disease, nor febrile symptoms enough to justify the enlargement being diagnosed as one of an inflammatory kind. While, on the other hand, the pain negatived the idea of hydatids, or amyloid or fatty degeneration, being the cause of the increased size of the organ.

To add still further to the difficulties in the way of diagnosis, the enlargement of the liver seemed to be general, which is seldom the case either in cancer, abscess, or hydatid disease, in all of which it is usually the right lobe of the organ alone that is affected. It did not look exactly like a case of impacted biliary concretion or calculus. (*a*) On account of there being but little acute pain, and (*b*) its advent having been gradual. (*c*) There being no itching of the skin. (*d*) There being intense prostration, and (*e*) increasing loss of flesh. A case presenting such a combination of anomalous signs and symptoms I had never up to that time seen. So I felt sorely puzzled to say what it really was.

The patient, being a man of mental acquirements, had given no ambiguous answers to my questions. On the contrary, he had given me the history of his case, and explained its symptoms, with unusual lucidity, as well as detailed the opinions of the different medical men he had consulted with a minuteness that sur-

prised me. From their all being of the opinion that it was an example of rapidly advancing malignant disease, and the absence of a cancerous cachexia making me doubt the correctness of that diagnosis, I hesitated in giving an opinion before I had analysed the urine. After summing up in my mind the respective values of all the negative as well as the positive signs and symptoms, I came, by a process of elimination, to the conclusion that the case could only be one of three things :—Either an anomalous case of cancer, an anomalous one of gall-stone, or an anomalous one of chronic hepatic congestion. I therefore told the patient that, although not prepared to say what the case really was, I felt almost certain that it was not one of malignant disease, and that I believed a properly conducted chemical analysis of his urine would in all probability reveal its true nature. For, as I explained to him, if the urine was found to contain bile-acids it would show that the jaundice was due to obstruction of the bile-ducts, and thereby prove the case not to be one of chronic congestion, but one either of cancer or gall-stones. While, again, the amount of uric acid eliminated in the twenty-four hours would probably in its turn decide between cancer and biliary concretion. The course for me to pursue was therefore plain enough—subject the urine to a careful chemical analysis, and diagnose according to its teachings.

It was soon arranged that he should send me samples of his urine, with the estimated quantity he passed in the twenty-four hours, and come and see me again that day week, when I hoped to be in a position to give him a definite answer. The urine arrived in due course, and was taken to University College for analysis. The analysis was made, and two most important facts were ascertained. First, it contained bile-acids, which, while it negatived the idea of chronic hepatitis, left it an open question whether the obstruction of the bile-duct was due to the presence of a cancer or a biliary concretion. Secondly, it contained little more than half the calculated normal amount of uric acid, which in its turn, while it almost negatived the idea of cancer, strongly favoured the idea of impacted biliary concretion. Acting then upon the principle that out of a multitude of conflicting data it is wise policy to accept the theory, however weak it may be, least open to objection, I consequently accepted that of impacted biliary concretion, attributing all the anomalous signs and symptoms presented by the case to the inconsistencies of the erratic course either of a fragment of inspissated bile or a true gall-stone.

Being armed with the above additional piece of chemical knowledge, I was now prepared to put some more specifically defining questions to the patient, with the view of eliciting, if possible, some clue to an

antecedent bilious history. They were put, and, the replies to them being all in the affirmative, I unhesitatingly told him that he laboured under no form of fatal disease whatever, but, on the contrary, one that was, barring accidents, perfectly curable. To my amazement he received this piece of information with anything but thankfulness, plainly telling me that he thought it was very wrong of me to attempt to raise futile hopes in the bosom of a man at his time of life, who had learned to look with calm resignation on a speedily approaching end, and it would be cruel of me if I attempted to excite in the minds of his wife and children anticipations which he instinctively felt could never be realised. Naturally enough this made me feel the gravity of the position I had assumed; but knowing that I had not assumed it without due consideration, I simply replied: 'It is not I that say what is the matter with you; it is science, and science is never wrong. Although I, as its interpreter, may be wrong, I have only said what I believe to be the truth, and I leave you to act upon it or not, just as you please.' A little more conversation, and it was arranged that, before my opinion was made known to the family, Mr. Jacob should be consulted. He was at once telegraphed for, and I had an interview with him the next day. After hearing the view I took of the case, and having explained to him the data upon which the opinion of

its being one of impacted biliary concretion was based, Mr. Jacob, of his own accord, said he would advise the patient to place himself at once unconditionally in my hands, and he hoped that the result would prove the accuracy of the diagnosis. From that day the patient put himself unreservedly under my care. The *fons et origo* of his malady was attacked: the obstruction in the common bile-duct was attempted to be removed, and with what success the sequel shows. Within six months from that day he was, comparatively speaking, well. Never since has he had a relapse, and now, after thirteen years have passed away, and he is seventy-four years of age, he is still in the full enjoyment of excellent health and spirits. I last saw him on July 10, 1880, when, being up in London, he paid me a friendly visit, and he then told me that he never had been in better health in his life, and most assuredly his looks appeared to confirm the assertion. For he looked both hale and hearty, and, as I said to him, likely enough to live for twenty years to come. Here, then, is another admirable living example of the benefits which accrue from the introduction of pure science into the practical domain of clinical medicine.

Seeing that the diagnosis of hepatic cancer is so difficult, I shall now put into a synoptical form a few additional hints, which, when taken in conjunction with what has already been said regarding its

symptomatology, may materially facilitate its detection.

Additional Hints to aid the Diagnosis of Hepatic Cancer.

1. As fully three-fourths of the cases of true hepatic cancer are the secondary result of a precisely similar pathological condition pre-existing in another part of the body—the stomach, the intestines, the mammary gland, the uterus, &c.—a knowledge of the nature of the morbid product co-existing in the other organs of the body will, as a rule, suffice to reveal the true nature of the disease affecting the liver. The one often being the mere prototype of the other. Be it colloid, scirrhus, melanoma, epithelioma, or what else it may. At the same time it must be remembered that, in accordance with the law of transmutability, cancers of different kinds may actually exist in the same patient at the same time.

2. When the nature of the co-existing morbid growth cannot be correctly determined, the existence of a well-marked cancerous cachexia, with enlargement of the abdominal, inguinal, axillary, or cervical glands, may be taken as evidence of the morbid hepatic deposit being encephaloid.

3. The palpable existence of nodules on the parietal surface of the liver favours the idea of the case being one of encephaloid disease. Though colloid is also occasionally nodulated.

4. If the disease progresses rapidly, the case may, almost for a certainty, be put down as one of encephaloid. For that is the form of hepatic cancer which is by far the most rapidly fatal.

5. If the so-called cancerous tumour is of slow growth, non-nodulated, hard, and resisting on pressure, and but slightly painful, it is most likely a non-malignant form of growth.

6. Primary hepatic cancer is frequently a true encephaloid.

7. When a pulpy, non-fluctuating tumour projecting from the surface of the liver can be distinctly felt, and other symptoms and signs of cancer are present, but without a cancerous cachexia, the case is most likely to be one of fungus hæmatodes.

8. When in doubt, to diagnose the case as one of encephaloid disease is wise policy. For as that form of cancer is by far the most common one affecting the liver, that opinion is the most likely to prove correct.

9. The enlargements and other derangements of the liver arising from multilocular hydatids, as well as from fatty and waxy disease of the hepatic tissue, have often been confounded with cases of cancer; but if the before-mentioned signs and symptoms are carefully considered, and if it is at the same time borne in mind that hydatid, fatty, and waxy enlargements are invariably painless, there is but little likelihood of even the novice confounding these affections.

10. While studying the diagnosis of a suspected case of hepatic cancer, it ought never to be forgotten that frequently the disease has been preceded, if not even, as I suspect it often is, induced, by the irritative effects of gall-stones, a blow, or chronic hepatitis. Hence the pre-existence of temporary jaundice with biliary colic can only be relied on as negativing the existence of cancer when there is a total absence of all cancerous cachexia.

11. In cases of cancer following upon chronically impacted gall-stones, the liver, though at first enlarged, becomes, in the long run, atrophied, and jaundice is in general a well-marked sign.

12. In cases in which cancer supervenes on an atrophied liver, the jaundiced condition of the skin may be well marked ; but I have never as yet met with one single case of hepatic cancer in which there has been severe jaundice associated with acute paroxysmal pain. I desire particular attention to be paid to this remark, as on more than one occasion I have seen not only an erroneous diagnosis result from a want of this knowledge, but, what may appear perhaps to be more extraordinary still, a false view to have been taken even of the pathological conditions met with at the autopsy. So important do I consider this point, both from a clinical and pathological point of view, that I shall here introduce the details of a typical case of the kind, which shows how the inflam-

matory agglutinated parts round the healed perforation caused by a gall-stone may be taken for and described as a 'scirrhus' by the gentlemen making the autopsy of the patient.

The case I shall relate is that of a patient aged 50, a poet of local reputation, as well as the holder of a public appointment in one of our great provincial towns, who, when I first saw him, was intensely jaundiced. The stools were pipeclay-coloured, the urine being at the same time loaded with bile. He told me that he suffered from acute pain, was sick, and had occasional rigors. With such characteristic signs and symptoms, I, of course, with scarcely more than a moment's thought, diagnosed the case as one of jaundice from obstruction of the common bile-duct by impacted gall-stone. I treated the case as such, and the patient slowly and gradually improved. The acute pain entirely disappeared, and the skin got less and less of a jaundiced tint.

At length I lost sight of the patient altogether, and the first thing I further learned of the case was from reading a long and complimentary obituary notice of the patient in the papers sent to me by his widow. This led to a correspondence with her, and my being informed that 'cancer' was the cause of death. To which I replied that as there had been a *post-mortem*, and I doubted the probability of cancer being the cause of the death, I should feel obliged if she would

forward to the medical man who made the *post-mortem* the letter which I enclosed. In which I stated my diagnosis to have been jaundice from obstruction to the common bile-duct from gall-stone.

Knowing, of course, that it was quite possible that since I had seen the patient an important change might have taken place in his condition, and that it was within the range of probabilities that the impacted gall-stone which was the cause of all his symptoms and signs at the time he was under my care might have induced the development of cancer, I put emphasis in my letter to the medical attendant on the fact of my doubting that cancer was the immediate cause of the patient's death. This I did from my knowing that :—(*a*) There had been, up till the time at least when I last saw him, not a trace of a cancerous cachexia. (*b*) The history of impacted gall-stone being exceptionally definite. (*c*) The existence of well-marked jaundice. Which facts, taken in conjunction with my being aware that all sorts of growths and tissue degenerations which lead to fatal results are often erroneously spoken of as 'cancerous,' when not a vestige of malignancy exists in them, made me be thus particular. My letter brought forth the following reply from Mr. W. O. Jones, of Bowden, dated May 11, 1881:—

'After you last saw Mr. —— he gradually improved. The jaundice entirely disappeared. The liver

diminished in size until it could only just be felt about an inch below the ribs. The gall-bladder, which had been so markedly prominent, became imperceptible to the touch, and his healthy colour returned. He went to his business as usual, gained flesh rapidly, and his appetite and digestion were excellent. This happy condition of affairs lasted about seven months. At the end of February the old symptoms returned somewhat suddenly, though without any severe pain. He had occasional shivering and vomiting, great prostration, deep jaundice, no bile in the excreta from the bowels, urine very high-coloured, containing large quantity of bile. My diagnosis was, as in the previous attack, impacted gall-stone. For the first three weeks bile appeared in the fæces about half-a-dozen times ; since then there was never any trace of it visible. The liver enlarged until it reached within a finger's breadth of the umbilicus. The gall-bladder became as prominent and hard as in the previous attack. There was never—although often searched for—any hard nodule felt in the liver. He gradually sank and died on April 26. I made a *post-mortem* examination on the following day, but only opened the abdomen. The gall-bladder and ducts were very much distended and adherent to surrounding structures ; the coats of the gall-bladder were a good deal thickened and of a dull white colour. The duct was twisted upon itself. On opening the gall-bladder,

a quantity of thin pale purulent fluid escaped. I passed my finger into the duct, and was surprised not to find any calculus. There was a contraction about half an inch from its opening into the duodenum, hard and gristly, and about half an inch in thickness, and completely closing the duct. I think there is no doubt that this was scirrhus, though I never saw a case of scirrhus of the ductus communis before. On the surface of the liver about a dozen small nodules like small marbles were seen, and one large one, as large as a small Tangerine orange; this was covered by the ribs. On section of these, they presented the appearance of medullary cancer. The liver was nutmeggy throughout, and all the bile had degenerated into thin purulent fluid similar to what the gall-bladder contained. I should very much like to know your opinion of the course of the disease. Surely a case of cancer could not have lasted all these years, and apparently become cured for a time. Is it possible a large gall-stone could have passed without giving rise to the usual agonising pain? Is it probable or possible that the thickening (scirrhus) was originally simply inflammatory thickening due to pressure of gall-stone (or even ulceration), that during the seven months of comparative health this went on contracting, that it became cancerous, and that the liver became secondarily affected?'

As is here seen, the details of the autopsy given

by Mr. Jones make the pathology of the case as clear as noonday.

(*a*) The cause of the patient's death was the permanent occlusion of the common bile-duct.

(*b*) The occlusion was the result of the inflammation which had been set up by the gall-stone which had perforated its way into and escaped unobserved by the intestines. The agglutinated mass not being a scirrhous growth, but the hard contracted and cicatrised tissues at and around what had been the seat of the gall-stone's perforation. (See pp. 625 and 855.)

(*c*) The small projections on the surface of the liver, thought to be medullary nodules, were (in all probability) the ends of dilated bile-ducts. As is frequently known to be the case in cases of permanent jaundice from stricture of the common bile-duct, as I have already explained at p. 786.

(*d*) Two things excluded the probability of cancer having been the cause of the jaundice. 1st, the presence of rigors and acute pain, and 2nd, its disappearing for no less than seven months, and then reappearing and continuing up till the patient's death. This is not the history of a case of jaundice from cancer at all, but one of perforating gall-stone and subsequent permanent occlusion of the duct after the passage of the stone. (See pp. 112 and 769.)

(*e*) The morbid anatomical conditions of gall-

bladder, twisted duct, and agglutinations, together with the described contraction about half an inch from the duodenal orifice of the bile-duct, all plainly indicate the course taken, as well as the subsequent effects induced, by a gall-stone that had ulcerated its way into the intestines. Further :—

(*f*) It was during the time that the ulcerated opening into the intestines remained pervious (that is to say immediately after the passing of the stone) that the patient's health improved, and the jaundiced tint of the skin disappeared, again to reappear on the complete cicatrisation of the ulcerated opening which killed the patient. Just in the same way as happened in the case the parts of which are represented in Plate I., p. 113.

Lastly, I think that I may venture to say from the result of experience that the signs and symptoms most often mistaken for cancer of the liver are those arising from chronically impacted biliary concretions. For even some of our most distinguished hospital physicians, to my personal knowledge, have fallen into this error. Which is not at all surprising, seeing that in both sets of affections there may be the common symptoms of pain, vomiting, jaundice, prostration, and emaciation. In some instances, indeed, the only way I know of distinguishing between the two classes of affections is to bear in mind that in the case of biliary concretions all the most marked signs and

symptoms are produced rapidly, whereas in cases of cancer they are, comparatively speaking, of slow development. For example, in cases of impacted biliary concretions—inspissated bile, or gall-stones—

a. The discoloration of the skin in most cases begins within seventy hours after the onset of the pain.

b. Pipeclay-coloured stools and dark urine are equally rapid in appearing.

c. The pain is not only acute, but accompanied, in the majority of instances, with rigors as well as vomiting.

d. The vomiting in cases of gall-stone seldom occurs without being associated with paroxysmal pain. Whereas in cases of cancer the pain is seldom or never paroxysmal.

e. The history of previous attacks of biliary colic, associated with clay-coloured stools and jaundice, taken in connection with a total absence of cancerous cachexia or enlargement of the glands, may, in the majority of instances, be regarded as conclusive evidence against the case being one of cancer.

Jaundice the Result of Cancerous Disease elsewhere than in the Liver.

The most frequent cause of jaundice in cases of hepatic cancer is said to be the direct result of the pressure of the cancerous mass upon the bile-ducts

blocking up their canals. I must, however, call attention to the fact that it is no uncommon thing for an occlusion of the common bile-duct to occur, not only from mere cancerous infiltration of its walls, but also from growths in their parietes, and that in both sets of cases a well-marked jaundice is produced. Likewise that cancer of the pyloric orifice of the stomach has been known to produce jaundice by obstruction in consequence of its extending to the walls of the common bile-duct, and thickening them to a sufficient extent to entirely occlude the passage of the bile. But even when the walls of the duct have not been attacked by the malignant deposit, jaundice has also been known to occur from the pressure exerted upon the orifice of the duct by a pyloric or a surrounding mass of duodenal cancer.

Pressure on the duct from a malignant growth in the lesser omentum, without the liver itself or its appendages having been affected by the disease, has also caused jaundice.

Dr. Bristowe has recorded[1] the case of a man aged 68, who suffered from jaundice, in whom the liver was small and the seat of scirrhous infiltration, and whose capsule of Glisson contained a few peritoneal cancerous nodules. The cause of the jaundice, however, was a scirrhous tumour of the lesser omentum, involving and constricting the walls of the common bile-duct.

[1] *Pathological Society's Transactions*, vol. xi. p. 127.

Cancer of the Liver may cause complete Occlusion of the Vena Cava Inferior.

A case of this kind is recorded by Dr. Little in the Dublin Pathological Society's 'Transactions,' 1878. The patient, a man 26 years of age, was only ill three months, and at the *post-mortem* the liver was found greatly enlarged, with deposits in it of spherical masses of primary cancer, which had caused such complete obstruction to the vena cava as to make the vena azygos assume the dimensions of the vena cava. There was a complete system of communication between epigastric, anterior intercostal, and internal mammary veins on the one hand, and iliac and thoracic veins on the other. Though the urine looked bilious, there was no distinct jaundice, and no ascites appeared until just at the last. The external veins of the trunk were distended and varicose, not only in front, but on the side of the body.

In a case of Cancer of the Liver, Death may occur from Hæmorrhage.

A case of death from hæmorrhage from a medullary cancer of the liver occurred in Middlesex Hospital in 1861. The patient, a man aged 50, under the care of Dr. Goodfellow, suffering from jaundice, was suddenly attacked with great prostration, urgent vomiting, distended abdomen, rapid pulse, great pain

and tenderness in the region of the liver. The next day he vomited a quantity of dark bloody-looking fluid. This was soon afterwards followed by collapse, which in a few hours ended in death.

At the *post-mortem* examination about six quarts of red bloody serum were found in the peritoneal cavity. While on the upper surface of the right lobe of the liver, and in contact with the diaphragm, was a coagulum weighing five ounces. Besides which the intestines were ' bathed with bloody fluid.' The liver weighed seventy-two ounces. The left lobe was atrophied to the appearance of a mere appendage of the right, not exceeding $1\frac{1}{2}$ inches in diameter transversely, and had a cirrhosed granulated-looking surface. The surface of the right lobe was studded over with prominent nodules, varying from the size of a pea to that of a cherry. The larger of the nodules were elastic, and the hæmorrhage was ascertained to have come from the bursting of one of them. In the interior of the liver were a number of cherry-sized cavities, filled with soft yellow cancer. The hæmorrhage was supposed to have begun three days before the patient's death.

Treatment of Hepatic Cancer.

Alas! for the credit of medical science, no cure has as yet been discovered for cancer. The physician's skill is unable to cope with it, for his pharmacopœia

contains no antidote. The surgeon cannot conquer it, as his knife fails to reach constitutional disease. To alleviate its discomforts, to retard its inevitably fatal progress, is therefore all that is within human power. Consequently, when once the case has been clearly diagnosed as one of cancer, all our efforts must be directed to foster the patient's vital powers, to increase his strength, and to relieve his sufferings. In this respect we have much within our scope, and fortunately for the credit of our profession we can even do more. For although we cannot eradicate the disease when once it has permeated the system, we have it in our power in many instances, even in the hereditarily predisposed, to ward off its attack. Things which of themselves may be regarded in the light of no small mercies, when it is considered that the babe in the cradle and the dotard on the verge of the tomb are alike liable to become the victims of the formidable affection we denominate cancer.

The lines of action which I recommend are as follows:—Having either detected the presence, or suspected the tendency to the development, of an hepatic cancer in a patient, an attempt ought to be at once made to stop the development of the local manifestation or control the growth of the disease. First, by improving the patient's constitution, and then maintaining it at as high a standard as is possible. How can this best be done? By diet by

regimen, and by the avoidance of everything which tends to exhaust the vital powers or impede the proper performance of the animal functions.

In order to do this successfully the patient's food must be of a mixed character. Flesh and fish, but neither salted nor high-seasoned. No frizzled bacon or salted haddock. No curries, sausages, or spiced meats. Let the flesh be that of mature animals, not of babes and sucklings, which is both indigestible and non-nutritious. Let the fish be sea-fish, but neither salmon nor mackerel, crabs nor lobsters. Allow a free supply of fresh vegetables and fruits, both cooked and uncooked. Let the vegetables be potatoes, cabbage, cauliflower, carrots, turnips, celery, lettuces, and watercress. Neither peas nor beans, for both are heating. No pastry, but milk and egg farinaceous puddings of all kinds. Corn-flour, rice, sago, arrowroot, tapioca, and such like. If stimulants are required, let them be those which possess nutritive qualities. Such as wines and beers. Not brandies or whiskies, which stimulate much, while they nourish little. Keep the skin clean, and in good acting condition, by a nocturnal and matutinal 'dry-bath,' in the shape of hair gloves, or skin scrubbing-brushes. Give plenty of opportunity for sleep, that is to say ten hours of bed. Avoid gymnastics and all possibility of receiving physical injury; for nothing is so conducive, as already shown, to

the local formation of a cancer in the predisposed as a blow or a bruise, from the fact that all local irritations lead to tissue degeneration in predisposed constitutions. Regulate the outdoor exercise according to the powers of the patient, by telling him or her to walk as much as he or she conveniently can without being fatigued. The moment that fatigue has been experienced it is a sign that too much has been done, and consequently harm instead of good. The sensation of fatigue being nature's cry for rest.

By a judicious and timely administration of remedies, relieve pain, promote sleep, and regulate the bowels. Prevention being always better than cure, the action of stomach, liver, and kidneys must be attended to with special care. Digestion favoured by stomachics, the biliary function by cholagogues, the action of the kidneys by diuretics. See that there are no deposits in the urine ; if there are, stop those of the acid variety by alkalies ; those of the alkaline and the earthy varieties by acids, on principles dictated by a mixture of scientific knowledge and common sense. Lastly, and not least, attention should be paid to keeping the patient cheerful, by diminishing as much as possible the cares of life. In fact, I generally embody my advice in the three following golden rules :—

Take amusement, without excitement ; exercise, without fatigue ; and nutrition, without stimulation.

CHAPTER XIX.

SYPHILITIC DISEASE OF THE LIVER.

Syphilitic disease usually attacks the liver in the form of well-defined, circumscribed nodules. The deposits vary in size, from small microscopic objects up to the dimensions of an orange, and present different naked-eye appearances according to their states of maturity. On section, a small pea-sized one has a distinctly reddish-white colour, while one, varying from the size of a cherry to that of a walnut, looks like a dirty yellowish-white mass, of moderately solid consistence. While, again, one as big as an orange has a decidedly yellow, cheesy, somewhat softened appearance.

All appear to the naked eye to be surrounded by a semi-translucent capsule, which, on close inspection, however, is seen to be merely the differently coloured outer margin of the mass, gradually shading away into the surrounding healthy liver tissue. So the nodules cannot be enucleated. When examined with the microscope, scrapings, as well as sections, are seen to be composed of a fine fibrous matrix, in

which are embedded fatty granular cells and albuminoid matter, very like what one sees in true tubercle. Among the débris cholesterin crystals are occasionally visible.

When the liver is injected, and the syphilitic nodules, after being hardened in absolute alcohol, examined under a microscope, they are found to be freely supplied with blood-vessels.

Syphilitic present marked anatomical differences from cancerous liver nodules. For example, they never project from the surface of the liver, but, on the contrary, are always imbedded in its substance, and, in consequence of the shrinking of the hepatic tissue, the portion of the liver over them is indented or even depressed. The large nodules show a great tendency to soften, but instead of softening into a creamy fluid, like medullary cancer, their softening resembles a purulent degeneration.

Etiology of Syphilitic Nodules.

a. They are almost always multiple.

b. When small, they may occur in great numbers. So many as fifty, and even a hundred, having been met with in one liver.

c. They are merely local manifestations of constitutional disease-germ impregnation.

d. They are a tertiary form of syphilitic disease, and are associated with the special form of cachexia—the so-called syphilitic cachexia.

e. They may occur at any age. The infant, as well as the adult, being liable to be affected with them.

f. They may be hereditary as well as acquired.

g. They frequently co-exist with similar growths in other parts of the body.

h. Dr. Wilks (who has given special attention to these forms of liver deposits) justly, I think, compares them to the peculiar fibrous forms of deposit met with in the tongue, intestinal canal, brain, muscles, and testes of patients labouring under tertiary syphilitic disease.

i. Syphilitic nodules are not unfrequently associated with tubercular deposits in the lungs, especially those of the miliary variety.

j. Moreover, we find that syphilitic nodules are also occasionally associated with amyloid degenerations. Not alone in the liver itself, but in other and distant organs of the body.

k. Not only may syphilitic disease of the liver be met with at all ages between the cradle and the coffin, but it may even have an intra-uterine origin. For a child came under Mr. Canton's care at the Charing Cross Hospital, which was scarcely seven weeks old when it died, and although the mother, a prostitute, aged 18, stated that she had never suffered from syphilis, the child had such distinctly copper-coloured syphilitic lepra spots over its body

as to leave no doubt in the mind of Mr. Canton that it was the subject of inherited syphilis. Moreover, on the *post-mortem* examination, no organic disease whatever was found in the body, except a number of fawn-coloured, putty-like deposits, varying in size from that of a pin's head to that of a pea, scattered irregularly throughout the liver. An excellent chromolithograph of their appearance is given in plate iv. vol. xiii. of the Pathological Society's 'Transactions.'

Symptoms of Syphilitic Liver.

Syphilitic disease of the liver only produces jaundice when it attacks the neighbourhood of the hepatic and common bile-ducts, and either implicates or compresses them to a sufficient extent to occlude their channels. Many continental physicians are, however, of opinion that the syphilitic taint itself is sufficient to induce jaundice, an opinion which I am not at all inclined to endorse; for although syphilitic patients are, as a rule, sallow, few, if any, of those I have ever met with could be said to be actually jaundiced. Moreover, if jaundice is a concomitant of syphilis (although no doubt it may be the direct result of the syphilitic poison), I am certainly more inclined to look upon it as the product of the mercury which has been given as a remedy for the disease. Mercury being one of the mineral poisons (as pointed

out in the special chapter upon the subject, p. 702) which produce jaundice. Syphilitic livers are seldom, if ever, appreciably increased in size. Hence we have not so much as that physical sign to aid us in their diagnosis. However, the absence of this sign does not much matter; for whenever an ill-conditioned, anæmic, dark, bilious, sallow-complexioned, cachectic-looking syphilitic-tainted individual complains of hepatic discomfort, with disordered biliary symptoms, for which no direct cause can be assigned, the case is in all probability one of tertiary syphilitic disease of the liver. And this supposition may be considered almost a certainty when tertiary syphilitic deposits are detectible in any other part of the body—as, for example, in the tongue or testes.

Treatment of Syphilitic Liver Disease.

The first step in the treatment of syphilitic disease of the liver is to improve the general health, and support the vital powers by administering every two hours small quantities of the most nourishing, non-stimulating kinds of food. Especially in the form of milk and egg farinaceous puddings, of which fortunately we have a large supply to select from, and can vary them from hour to hour. For example, there are arrowroot, tapioca, sago, semolina, corn flour, revalenta arabica, rice, oatmeal, and a host of others well known to every housewife, who in this

instance must act as the handmaiden of the doctor. Besides these farinaceous foods, animal and vegetable broths are to be freely administered, ox-tail and hare soups, rabbit, chicken, and neck-of-mutton broths. All kinds of white fish may also be given in moderation. But neither eels, salmon, nor mackerel, which of our native kinds of fish are the most liable to disorder the digestive and hepatic functions. Moreover, all foods that have undergone the process of salting, as well as all tinned foods, are to be eschewed as being only moderately nutritious, while they are at the same time less easy of digestion.

No beer or wine is, as a rule, to be given ; but of course where in any exceptional case either or both seem desirable, they are not to be forbidden. Then as regards pharmacopœial remedies, we have in the anti-syphilitic category a whole host to select from. Mercurials, however, must in the majority of cases be left aside, and iodides and bromides employed in their stead. And as regards them, I may remark that I prefer, in the case of hepatic syphilitic disease, to give the patient ammonium iodides and bromides instead of potassium ones, on account of their being less lowering and more hypnotic. Lastly, careful attention to ordinary sanitary laws is indispensable, cleanliness being in this case an invaluable aid to treatment. Early to bed and early to rise is to be strictly enjoined.

Benign Forms of Hepatic Disease.

The so-called benign forms of hepatic disease are not to be regarded as benign in the sense that 'they do not kill;' for, on the contrary, some of them at least are inevitably fatal affections. Their so-called benignness consists in the fact that they are not painful affections, and give rise to very little constitutional disturbance. Under the heading of benign affections of the liver regarded in this sense may be included hydatids, fatty, amyloid, and fibrous degenerations, simple cysts, emboli, and blood extravasations. Even a so-called fibrous degeneration, which, in contradistinction to forms of disease productive of grave constitutional symptoms, might be regarded as the type of a benign liver affection, is yet wholly beyond the powers of the healing art. For though producing but little discomfort to the patient, it slowly marches on to a fatal termination, with as steady and as relentless a pace as the most rapid form of cancerous degeneration. None of these so-called benign forms of hepatic disease is necessarily associated with jaundice. Rarely indeed does the skin present more than a sallow appearance, while saffron-coloured urine is not often met with, and pipeclay stools rarely or never seen.

CHAPTER XX.

HYDATID DISEASE OF THE LIVER.

This disease resembles, in its medical history, cancer of the liver in four respects. Firstly in being not quite so uncommon a complaint in this country as the general run of medical men who have not made liver affections a special study, appear to imagine. Secondly, from its being one of those hepatic affections with which jaundice is by no means necessarily, or even so much as usually associated. Thirdly, in generally attacking the right lobe of the liver alone. Lastly, in occurring at all ages.

Etiology of Hepatic Hydatids.

Although all my readers are of course perfectly aware that a hydatid of the liver is an animal parasite, possibly some of them may not be equally aware of the fact that the parasite is nothing more or less than an early and transitional stage in the life's history and strangely varied developmental career of a dog's tapeworm. A species of entozoon

also found in the intestines of the fox and the wolf. So it may well be asked, ' How does this parasite get from these animals into the human liver ? ' I reply, easily enough, and most probably in the following wise. A dog infested by a tapeworm passes thousands, nay, probably millions, of the parasite's eggs along with its fæces. The fæces becoming dry crumble into dust, to which dust the eggs of the tapeworm tenaciously adhere, the first gust of wind whisks them into the air, and they get blown about here and there and everywhere. At last some of them land in a fruit or vegetable garden, where they attach themselves to any ripe or unripe fruits and vegetables which happen to be in their way. Such as strawberries, gooseberries, apples, pears, lettuces, radishes, celery, watercresses, and all the other kinds of garden produce which are daily sold in our markets and hawked about in our streets, and which men, women, and children, rich and poor alike, often eat in an unwashed state, swallowing at the same time all the worm-eggs which happen to adhere to them. A number, however, are not required to produce a hydatid in the human liver. A single one is sufficient for that purpose—ay, even to generate a thousand hydatids. For so soon as it finds a suitable habitat in the human body, it hatches, develops, and multiplies with rapidity. No wonder then that hydatid disease is so common amongst us. The

marvel to me is that it is not more common still.

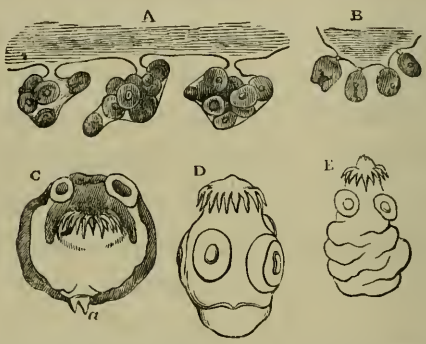
I may begin my description of the disease by making a few remarks upon the nature and life's history of the parasite which gives rise to it, the scientific name for which is *Echinococcus hominis*. An entozoon which differs considerably in its mode of action upon the human body from many other forms of entozoa. For in the first place it cannot develop and arrive at maturity in the body of the individual in which it is born. It cannot so much even as advance beyond the embryo stage until it has changed not only its habitat but its host. Then, and then only, does it attain to maturity, and become capable of propagating its kind by the production of eggs. Which, as I have just said, after being expelled from the intestines of their primary host, are subsequently conveyed by various means into another organism, where they develop into the larval form, and after a time become hydatids. In their turn again to change their host, in some cases returning even into the species of animal from which the eggs originally came, and then become mature animals—tapeworms. This process of change is characterised by the term 'alternate generation,' and in describing it authors make use of the terms *Strobila*, to signify the entire worm, sexually mature; *Proglottis*, the mature segment or joint; *Scolex*, the hydatid or larval form, which includes

the tapeworm head ; and *Proscolex*, the embryo contained in the ovum.[1]

The subjoined woodcut conveys some idea of the form and development of the scolices within the parent cyst as they are met with in the human liver.

The echinococcus cysts, which are developed from the six-hooked embryo of the mature Tænia, occur in

FIG. 27.



Echinococcus hominis (from Wilson). A, B. Grouped and single Echinococci, attached by peduncles to the inner membrane of the cyst. C. An expanded (*a*. The peduncle), and D, a contracted Echinococcus. E. A shrivelled animal.

various organs besides the liver. Even the lungs, kidneys, bladder, bones, neck, and several other parts of the human body are infested with them.

In a case in University College Hospital, the fluid taken from an apparently serous cyst in a woman's neck, I found, on microscopic examination, contained a number of echinococci, thus proving the tumour to

[1] See Dr. Cobbold's excellent work on Entozoa, published by Messrs. Churchill.

be an animal parasitic hydatid instead of an ordinary serous sac for which it had been mistaken and treated.

Hydatids are sometimes voided entire from the kidneys along with the urine.[1] Echinococcus cysts are often found in the liver of the horse and ox. Sometimes too in such numbers that scarcely any of the gland structure remains.

When a man, a sheep, or any other animal swallows the ova, their shells are dissolved by the digestive juices, and the tiny coiled-up six-hooked embryos escape. They then bore their way through the coats of the digestive canal, and getting into the blood-vessels are floated along with the circulating blood to the liver, or some other equally appropriate organ, in which they find a house, or habitat, as it is in scientific language called. No sooner have they found an appropriate resting-place wherein to take up their abode than they at once settle themselves down, as it were, and immediately begin to grow, and very soon develop into what is known to us under the name of hydatids. Even within three months or so from their settling down in their new homes, heads, with hooklets upon them, begin to make their appearance, and at the same time daughter, and shortly afterwards grand-daughter, vesicles are formed within the parent hydatid by a process of endogenous development.

[1] *Medical Times and Gazette*, March 25, 1865.

From the foregoing brief sketch of the parasite's life's history in so far as it relates to the human liver, it will be readily perceived how easy it is for a person to become the victim of hydatid diseases by swallowing along with his food a proscolex from a dog's tapeworm. It being almost certain sooner or later to develop itself into a hydatid. Curiously enough, it appears that the right lobe of the liver is of all places in the human body the one the parasite is most prone to select for its habitat during the cystic stage of its development.

Sometimes hydatids, developed in the hepatic organ, multiply, and spread themselves over the whole abdominal cavity, attaching themselves to the peritoneum, and penetrating into its substructures, be they intestinal, diaphragmatic, splenic, or renal. In such cases the parasites may not only be numbered by dozens, or even hundreds, but by thousands.

Dr. Gibb recorded a remarkable case of an enormous tumour of multiple hydatids 'springing from the anterior of the left lobe of the liver . . . invading every part of the abdominal cavity, becoming incorporated with the neighbouring structures, and especially with the entire peritoneal surface, penetrating it in front, at the sides, and upwards, the hydatids being attached to the muscular structure of the diaphragm and the abdominal muscles.' . . . 'There were probably some thousands of the animals,' and

they destroyed life by arresting (by their compression) its most ordinary functions.' The patient was a lad 16 years of age. (Pathological Society's 'Transactions,' vol. xvi. p. 159.)

In examining patients suffering from hepatic hydatids, I have been particularly struck with the fact that in general they trace back the origin of their disease to the reception of a local injury—a blow or a squeeze on or in the neighbourhood of the liver. Of course this cannot possibly be the originating cause of the animal hydatid, but is merely the reason of their attention having been called to its existence. The injury to the liver, or it may be perhaps to the parasite itself, producing sufficient inflammatory action and consequent local discomfort to call the patient's attention to it.

Signs and Symptoms of Hepatic Hydatids.

The chief points to be borne in mind in ascertaining the presence or absence of a hydatid of the liver in any suspicious case are the following :—

Hydatid disease may occur in children from two years of age and upwards. I may even say that, like many other diseases of the liver, it may have an intra-uterine origin. For Cruveilhier says an infant, only a few days old, was brought to him with a well-developed hydatid of the liver, which must have had an intra-uterine origin of some standing, as the cyst,

he tells us, burst spontaneously into the intestines as early as the twelfth day after the child's birth.

The parasite, from some as yet inexplicable cause, in nine out of every ten instances locates itself in the right lobe of the liver. The growth of the parasite is so insidious and slow, that it seldom attracts the patient's attention until it begins to cause discomfort from the pressure it exercises by its mere bulk on the neighbouring organs. And even then it is, as a rule, regarded, from the inconvenience it causes being but trifling, as a mere harmless swelling. By the time the medical attendant is consulted, the cyst has usually attained a sufficient size to appear as a visible projection from beneath the false ribs. I once made the *post-mortem* of a married woman, in University College Hospital, of 30 years of age, who, as far as I am aware, had never had an hepatic symptom in her whole life, and yet in the right lobe of her liver I found a hydatid cyst as big as a new-born child's head.

The tumour is usually perfectly painless, even on the application of firm manual pressure. But there are exceptions to this rule not generally recognised in books, to which it behoves me to call special attention, in order to prevent the possibility of the practitioner committing a grave error in diagnosis. Some hepatic hydatid cysts have been brought under the notice of the medical attendant solely on account of their being painful. The most important case of this

kind, in a clinical point of view, with which I am acquainted, is one that was published by Dr. Risel in the 'Deutsche Zeitschrift,' No. 45, 1874, from which, on its being opened by a direct incision, nearly seventy ounces of offensively smelling, bile-tinged, purulent fluid was withdrawn.

The pain in such cases is not due to the pressure of the hydatid itself, but to its being inflamed, and all its surroundings sympathising with the inflamed condition of the cyst. Had the hydatid in Dr. Risel's case been in a normal healthy state, I do not believe it would under any circumstances have been sensitive to pressure. In making a differential diagnosis in an obscure case of hydatid disease, it will be well, therefore, to take into consideration the possibility of its being (under certain unusual abnormal conditions) tender to pressure. A hydatid tumour may be either soft, and give rise to a distinct feeling of fluctuation, or as hard and as resisting as a stone. In order to save repetition I may refer the reader to a most remarkable example of this kind related at page 991, which I saw in consultation with Dr. Macaldin.

If the hydatid be in close proximity to the abdominal walls, and the patient not corpulent, when the left hand is laid flat and firmly on the tumour, and smartly tapped upon by the knuckles of the right (the hands being thus made to act the part of a pleximeter and hammer), a peculiarly tremulous

vibratory thrill—called the hydatid fremitus—is felt by the palm of the left hand, as it lies flatly and firmly pressed against the abdominal wall over the cyst. I am sorry to say, however, that this, which is otherwise a crucial and consequently a most important diagnostic sign, is far more frequently conspicuous by its absence than by its presence.

After having made this remark I rejoice at being able to add that, fortunately alike for patient and practitioner, the existence, in any given case, of a combination of any three of the above-named signs is in by far the majority of instances quite sufficient to lead to a correct diagnosis, if they are at the same time associated with an entire absence of distressing constitutional symptoms, and this, too, even when jaundice is present, as well as when it is absent. Unfortunately, however, as we shall presently see, all cases of hydatid disease of the liver are not equally easy of diagnosis. Some indeed baffle the skill of men of undoubted talent, and their consideration will specially engage attention a little further on. Meanwhile we shall consider the subject of

Jaundice from Hydatid Disease.

Jaundice from hydatid disease is far from common, about ninety per cent. of the cases being without it. When it does occur it is caused either by the external pressure of a cyst obstructing the outflow of bile into

the intestines from the common bile-duct, or what is more often the case—though still rare—from the hepatic duct being plugged up by hydatids. This occurs, I have little doubt, much oftener than is imagined, from the fact that when the hydatids are discharged into the intestines the jaundice disappears, the patient gets well, and no opportunity is afforded of ascertaining the cause of the jaundice or verifying the diagnosis, even when correctly made. It is only in rare cases, when the cysts are detected in the stool or the obstruction is permanent, and the patient succumbs, that we can positively ascribe the cause of the jaundice to the blocking up of the common bile-duct by hydatids.

Jaundice from the bursting of a hydatid cyst into the bile-duct is, I believe, by no means an uncommon occurrence. For several cases have been reported, both in this country and abroad, where hydatids, or portions of hydatids, have been discharged with, and discovered in, the patient's stools, and their discharge having been followed by a sudden cessation of the jaundice and biliary colic with which it was accompanied, has made the cases look like those of jaundice from gall-stones. Some of these cases unfortunately prove fatal, like one I shall presently relate that I saw along with Dr. Nuttall. (See page 981.)

Jaundice from Suppurating Hydatids.

An unusual case of jaundice from a suppurating hydatid, where bile flowed out along with the pus on its being tapped, occurred under the care of Dr. Ramskill at the London Hospital in August 1873. The case was that of a Londoner who had during five years been subject to what he called 'ague.' He had usually had two attacks every year, each continuing about a fortnight, which always commenced with cold sweating, on alternate mornings, between eleven and twelve o'clock. He had been jaundiced about a month. About the same time that he noticed the change in his colour, he found a lump growing in his right side, which gave him no pain.

There was a tumour coming down from under the right ribs, and extending below the umbilicus, and a little to the left of the median line. During respiration it protruded, but did not descend. The mass was soft, very elastic, and indistinctly fluctuating. The patient was very prostrate; his voice was reduced to a whisper; he seemed to be unable to move; his temperature was 103; pulse, 120; and respirations, 36.

The tumour was punctured, the pneumatic aspirator being used. Owing, however, to the canula becoming almost immediately plugged, only a few drops of thick offensive greenish fluid were evacu-

ated ; this, upon microscopic examination, showed a quantity of broken-up membrane, but no hooklets. A free opening was made with a scalpel through the abdominal wall about two inches above and one inch to the right of the umbilicus, and the depth of the wound was cautiously increased, until the whole of the blade of the scalpel had disappeared in the wound, when a free flow of purulent matter took place, to the extent of fully sixty ounces. A drainage-tube was left in the wound, from which a discharge, which often appeared to be only bile, continued for some time. The patient expressed his sense of great relief soon after the operation. About a fortnight afterwards, the wound threatened to heal ; a tent was, therefore, put in, and on examining it, a probe passed in to the depth of fully six inches. The discharge continued, to the extent of about two ounces daily of creamy-looking pus mixed with blood. Three months after his admission his colour was natural ; he had gained considerably in weight ; the discharge had ceased, and the wound apparently closed, and the patient expressed himself as being 'very well.' This is a case well worthy of being put on permanent record as an encouragement to operate even in apparently very bad cases.

Dr. Wilks has recorded [1] the case of a man aged 50, who died from an attack of jaundice, in whom

[1] *Pathological Society's Transactions*, vol. xi. p. 128.

was found, at the *post-mortem*, a large suppurating hydatid cyst of the liver, which had opened into an hepatic duct.

The Amount of Jaundice is not in proportion to the Size of the Hydatids.

That even very small hydatid cysts occasionally give rise to intense jaundice, is proved by the case related by the late Dr. Murchison, who had under his care, in the spring of 1869, a man, aged 34, with two small fluctuating and distinct tumours in the right hypochondrium. From the first five, and from the second seven, ounces of characteristic hydatid fluid were let out; and although the cysts were small, yet the man was deeply jaundiced, evidently from pressure upon the bile-duct. After tapping, the jaundiced tint of the skin disappeared, thereby confirming the correctness of the diagnosis, that it had been the result of the pressure of the distended cyst on the external wall of the bile-duct.

A somewhat anomalous case of supposed 'compound suppurating hydatid of the liver' is reported by Dr. Cockle and Mr. Rose in the 'Lancet' of July 8, 1882. It is briefly as follows:—A man aged 37, who had suffered from lumbago and loss of appetite for some time, eventually observed that his feet and ankles were swollen at night, and, having caught cold, had shiverings, recurring at irregular intervals, with severe cough and difficulty of breath-

ing. He had also pain in the stomach, and the area of hepatic dulness extended from the fourth rib in the right mammary line to two inches below the umbilicus. There was no jaundice. On December 2 there was a visible prominence over the middle of the enlargement. It was tense, elastic, with obscure hydatid fremitus on percussion, and a distinct feeling of fluctuation. Upon firm pressure over the prominence the fluid in the swelling seemed to be displaced, reappearing slowly with a rebound as the pressure was gradually relaxed. These signs led to the diagnosis of a compound suppurating hydatid cyst of the liver.

Mr. Rose opened the cyst by incision, on December 6, under the antiseptic spray. An incision one inch and a half in length was made just internal to the cartilage of the ninth rib ; a large amount of pus and about five hundred hydatids escaped. They varied in size from an ordinary pea to two inches in diameter. A large-sized caoutchouc drainage-tube was inserted. On several occasions during the progress of the treatment discharge of bile took place through the tube, and several of the cyst membranes were stained with bile. In many of the secondary cysts minute buds studded the germinal membrane. Such of the undamaged cysts as were microscopically examined contained hooklets. Some cysts were passed ruptured and decomposing. A stercoraceous odour, tolerably

persistent, was more or less detectible in the discharge through the tube, yet at no period did there exist the slightest ground for supposing that any communication with the bowel existed. By May 2, the patient was in excellent health, and able to resume his duty as a constable.

The reason why jaundice is nearly, though not quite, as rare in cases of hepatic hydatid disease as it is in cancer, is that it can only arise in those exceptional cases where the parasite, like the cancer, by its position causes an actual mechanical obstruction to the outflow of bile through the common bile-duct, either by exerting pressure upon it directly from without, or from hydatid vesicles gaining access to its interior, and plugging it up.

Diseases which simulate Hydatids of the Liver.

As there are several forms of disease which closely simulate the physical signs of hepatic hydatids, before leaving the symptomatology it will be well for me to specially allude to them. These diseases are :—A dilated stomach, a distended gall-bladder, a hydronephrosis, an ovarian tumour, pregnancy, a phantom tumour, encephaloid disease of the liver, an abscess, impacted biliary concretions, and cystic disease.

An Hepatic Hydatid may simulate a Dilated Stomach.

The best example I can cite of an hepatic hydatid being mistaken for a dilated stomach is a case I saw along with Mr. Beale of Paddington, in 1865. The patient was a lady aged 27, who had been married three years, but never been pregnant, and never ailed anything until eleven months before I saw her, when she began to feel distension and gastric discomfort after eating. Oftentimes to such an extent that she had to loosen her stays. At the same time she noticed that her skin was assuming a more and more dusky hue. Two months before our consultation, she for the first time observed that her stomach projected visibly from beneath the breast-bone, and that it was impossible for her any longer to wear her ordinary stays. She then called in Mr. Beale, who brought her to me, under the impression, in consequence of her gastric symptoms, that she was suffering from a dilated stomach. On examining her very carefully, I found that there was a distinct fulness and bulging of the abdominal parietes just below and more particularly to the right of the xiphoid cartilage, and that, instead of her measuring round the waist 20 inches, as she said she had done a few months before, her girth was 27 inches. The liver dulness in the perpendicular nipple line was ascertained to be 5½ inches. Her skin, though very

sallow, was not sufficiently so to merit the title of being jaundiced. The stools were light, though not pipeclay-coloured, and the urine a dark brown sherry-wine colour, and containing a copious sediment of urates. On manipulating the swelling carefully and percussing it, I detected a vibratory fremitus, which sign instantly settled the diagnosis. The swelling was not a dilated stomach, but an hepatic hydatid. Tapping was proposed, but the patient would not submit to it. The case, I was told, ultimately ended in a spontaneous cure.

Liver Hydatids mistaken for Ovarian Cysts, &c.

That an hepatic parasite may be fatally mistaken for an ovarian tumour, and operated upon as such, is well shown in a case recorded by Dr. Ward Cousins. A woman aged 27, who, on admission into the Portsmouth Hospital, complained of shortness of breath, though her lungs and heart were quite healthy, when examined in the erect posture had the appearance of being at the full term of pregnancy. The outline of the tumour was somewhat irregular, the right side, especially above the umbilicus, being more prominent than the left. Entire dulness was elicited over the whole abdomen, except just below the margin of the ribs. Fluctuation was coincident with the dulness, and could be detected in all directions. Dr. Cousins diagnosed ovarian tumour, probably unilocular.

On January 7, 1874, he performed ovariotomy. An incision was commenced two inches below the umbilicus, and continued downwards for five inches in the median line. Slight retching occurred, which projected the tumour against the wound, and it appeared to move freely under the peritoneum; but, when this was divided, the cyst was found everywhere adherent by long bands to the parietes. The tumour was then tapped, and two gallons and a half of clear yellowish limpid fluid were withdrawn; the final portion which escaped was slightly opaque. The adhesions, which were almost universal over the front of the cyst, were readily broken down with the hand, and the mass detached from the pelvic brim. There was scarcely any hæmorrhage during the operation. The wound was closed in the ordinary way. The patient never rallied from the shock of the operation. Obstinate vomiting supervened, and she died in thirty-six hours.

Mistakes of this kind, although not often publicly recorded, are by no means uncommon. I shall, therefore, cite another equally important one that was published by Mr. Thomas Smith in the 'British Medical Journal,' February 1, 1868, where a hydatid of the liver, from which bile escaped during the operation, was attempted to be removed under the impression that it was an ovarian tumour. Mr. Smith says :—'A widow aged 39, who eight years before had

her last child, and immediately afterwards noticed a small firm swelling just above the left groin. This increased in size without causing pain until a year ago. Since then she suffered at times severely. The swelling increased quickly in size. On June 4, 1867, the abdomen was symmetrically distended. Fluctuation was very perceptible over the whole surface, which was dull to percussion ; the only resonant parts of the abdomen being the flanks, the epigastrium, and the hypochondria. There were in the abdominal walls one or two hard knots, which seemed to be unconnected with the tumour. The uterus was pushed very low down in the pelvis ; its fundus was plainly to be felt, and could be moved from side to side freely with the finger.

From the history of the case, and physical examination, little doubt existed that she was suffering from an ovarian cyst, probably unilocular. On July 11, the operation of ovariotomy was undertaken. An incision about three inches in length was made in the linea alba. On dissecting through the abdominal wall, a granular and adherent membrane, distended with fluid, was reached. This was opened with a minute puncture ; and, as fluid resembling that found in ovarian disease escaped, the separation of the cyst from the abdominal wall was attempted. This separation had been accomplished to some extent, when suddenly the cyst-wall gave way at the point of

puncture, and a hydatid escaped. The nature of the disease being now evident, the opening was enlarged, and an immense quantity of hydatids were discharged. They were of all sizes, from the size of a marble to that of a cocoa-nut. In all, eight pints of fluid escaped, and seven pints of hydatid cysts. The cavity having been emptied, the cyst-walls were drawn out and attached by silver sutures to the margins of the external wound.

The subsequent progress of the case was thoroughly satisfactory. The cyst was syringed twice daily with a weak solution of iodine (two drachms of the compound tincture to a pint of water). Occasionally collapsed hydatids escaped after syringing. At no time was there any disagreeable odour from the wound. The fluid that escaped was tinged by bile.

On September 10, the patient left in good health, with the wound soundly healed.

This case serves to illustrate the uncertainty of the diagnosis of abdominal tumours. Though the cyst must have been of hepatic origin, yet its connections, so far as could be ascertained, before, during, and after the operation, were chiefly among the lower part of the abdominal and pelvic regions. Nor is it peculiarly unique that a liver hydatid should present in the pelvis.

Hydatids simulating Pregnancy.

In the 'Medical Times and Gazette' of August 1864 is a case related by Dr. Sadler where Cæsarean section was undertaken at the full period of gestation. The tumour, which had been thought to be a pregnant uterus, was found at the *post-mortem* examination to be a calcified hydatid of the liver.

The stethoscope is the instrument which ought in these cases to be relied upon in making a differential diagnosis. For with a sharp ear the fœtal heart in a not very fat patient is detectible at the third month. I have, while house surgeon to the Royal Maternity, Edinburgh, occasionally even heard it as early as ten weeks after the termination of the menstruation.

Hydatids simulating Cystic Disease of the Liver.

In addition to ovarian there is another form of cystic disease which is not unfrequently mistaken for hydatids, and that too even after the death of the patient and the autopsy has revealed—or I should perhaps rather say might have been expected to reveal—the true state of matters. I allude to a form of affection which has received the title of 'Cystic Disease of the Liver.' So many are the errors committed in these cases that I shall subsequently devote a few special paragraphs to their consideration. Meanwhile I may mention that an exceedingly interesting case of hydatid disease was published under the above

title in the Pathological Society's 'Transactions' of 1857, p. 245, by Mr. Jabez Hogg, which has so many interesting features connected with it that I shall not only briefly quote it, but at the same time introduce its instructive illustrative woodcut (fig. 28). The patient, a man aged 45, from being jaundiced, and at the same time suffering from acute hepatic colic, with pipeclay-coloured stools and saffron-tinted

FIG. 28.

a, Cyst with echinoccocus within it. *b*, Hooklets of the entozoon. *c*, Crystals of cholesterin in plates. The true nature of the needle-shaped crystals is uncertain. They were all distributed irregularly through the hydatid fluid. *d*, Cylindrical epithelium, some enclosed in a structureless membrane. *e*, Puro-mucous corpuscles from outside of the cyst-wall. Mag. 200 diameters.

urine, was naturally enough supposed to be labouring under the effects of an impacted gall-stone.

After a time his symptoms assumed an alarming character. He had rigors, the lower extremities became œdematous from the feet to the loins. The superficial abdominal veins became enlarged, and he died suffering from distressing dyspnœa. On *post-mortem* examination the abdomen was found to con-

tain about a gallon of deep yellow-coloured serum. The liver was enormously enlarged, occupying the whole epigastric and right hypochondriac regions. It weighed eight and a half pounds. The right lobe contained a large hydatid filled with a gelatinous pale amber-coloured, thick, purulent, gruel-like, offensive-smelling fluid, which, when examined with the microscope, showed the elements represented in fig. 28.

It may be added, as points of further interest, that the hydatid had, by its external pressure on the bile-duct, completely obliterated its canal; that the left lobe of the liver was (most probably also on account of the pressure exercised upon its vessels and parenchyma) reduced to a softened mass, and its ducts charged with inspissated bile, while the gall-bladder contained four gall-stones.

Hydatid Disease may be mistaken for Cancer of the Liver.

In consequence of hydatids sometimes appearing on the free anterior surface of the liver in a multiple form, and not exceeding the size of a small orange, they have been by the unwary mistaken for, and treated as, encephaloid tumours. This is readily accounted for by the fact that the feeling they give rise to through the abdominal parietes is identical with that communicated to the hand by cancerous nodules. While in addition they still further simulate cancer in

being usually situated in the right lobe, of slow growth, and the discomfort caused by their presence, amounting occasionally to distinct pain. These cases are, however, with proper care usually easily differentiated, from the fact of the most prominent differential characteristic features between these two forms of liver disease being :—

a. The total absence of constitutional symptoms in hydatid disease.

b. An entire absence of a cancerous cachexia.

c. The nodules usually not being painful on pressure.

The Jaundice arising from Hydatids may closely simulate that from Gall-stones.

In the Pathological Society's 'Transactions' (vol. xxvi. p. 127), Dr. Cayley records the case of a labourer, aged 36, who was occasionally seized with violent pain and vomiting, followed by jaundice, which would last a few days and then pass off. Twelve of these attacks occurred in a year, and at the last a number of hydatid vesicles the size of peas and beans were found in the stools. After which he got quite well. (See also case at p. 981.)

When a hydatid bursts into the common bile-duct, the pain produced by the cyst vesicles is so similar to that arising from an impacted gall-stone as to be in general mistaken for it, and it is only when the

vesicles, or portions of them, begin to pass by the stools that the true nature of the attack is revealed. For the blocking up of the duct by the hydatid gives rise to jaundice, pipeclay-coloured stools, and high-coloured urine, pain, itchiness of the skin, &c., exactly in the same manner as a gall-stone or a mass of inspissated bile.

Hints on the Differential Diagnosis of Hepatic Hydatid Disease.

a. Jaundice will most likely be associated with a distended gall-bladder.

b. Pain and febrile disturbance always attend the presence of an abscess, and the swelling is usually acutely tender on pressure, which is never the case in a non-inflamed hydatid.

c. The mobility of an ovarian tumour, together with its history and the result of a vaginal examination, usually suffices to distinguish it from a liver hydatid. The hepatic hydatid has its dull area distinctly continuous with that of the liver, while the ovarian tumour very seldom has. An ovarian tumour is more or less mobile under a uterine sound examination, while a liver hydatid never is.

d. In the case of a dilated stomach there is usually a specific history of gastric derangement, which is seldom the case in hepatic hydatid.

e. In the case of hydronephrosis there is almost

sure to be a distinct history of a urinary derangement having existed for some length of time.

f. A phantom abdominal tumour vanishes at once when the patient is placed under the influence of an anæsthetic, to return immediately he or she becomes again conscious.

g. As, notwithstanding all these apparently so easily recognised diagnostic beacons and readily applied differentiating tests, there yet oftentimes lurk beneath the surface of the seemingly most simple cases conditions, ignorance of which has led even experienced men into grave errors in the differential diagnosis of hepatic hydatids, I shall now direct attention to what I look upon as being my best friend in doubtful cases. Namely, the exploring needle, an instrument far too much neglected by physicians, as I have already observed, under the mistaken idea that its employment is not altogether free from risk. I have never once seen the exploring needle do harm, although I have used it under circumstances which, were I to relate some of them, would make those unaccustomed to its use open their eyes wide with astonishment. I say, and say advisedly, that, when judiciously employed, the exploring needle is harmless, and in the case of a hydatid its employment will not only be the certain means of making an accurate diagnosis, but probably at the same time (if the fluid be allowed to drain away) effect a permanent cure of the dis-

ease without the necessity of any further treatment whatever. Emphatically, therefore, do I recommend the employment of the exploring needle in every case of doubt, no matter whether the tumour be seemingly distinctly attached to the liver or not. For, as I shall immediately show, an examination of the fluid withdrawn when it contains fluid, or of the tissue brought away when it is solid, will, if properly performed, never fail to reveal the true nature of the suspected tumour.

After making these remarks I think it my duty to point out

How to Explore a Hydatid.

I may remind the reader of what I said in the chapter on abscesses ; namely, that a human liver may be penetrated by an exploring needle in all directions without danger. But as there is a right and a wrong way of doing everything, even the very simplest act, it is well for me to give a few hints as to the best way of doing it.

Begin by marking out with ink the exact *absolute* dull area, after which select the most prominent part of it for the first puncture. After having lubricated the needle with carbolised oil, push it in rapidly and with some force from three to six inches into the tumour. Withdraw the trocar and allow the canula to remain quietly in its position for at least thirty

seconds, in order to give fluid, if it chance to be thick, time to escape. If nothing comes away, then slowly and gradually withdraw it, pausing at every half-inch of its withdrawal, in the hope that (should the cyst be a small one and the exploring needle have entirely transfixed it) the end of the canula during the time of its withdrawal may get within the confines of the sac, and its fluid contents thereupon escape.

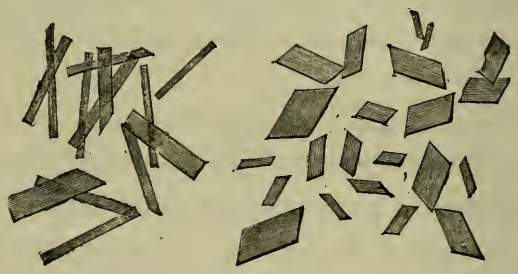
Should the first puncture be attended with negative results, let the operation be repeated at places about two inches apart as often as is thought necessary.

The Examination of Withdrawn Fluids.

Having been fortunate enough to withdraw fluid, the next thing is to be able to recognise its true nature. If it be pure pus or bile, there can be no difficulty about the matter ; but if the fluid be from a hydatid, an ovarian cyst, a hydronephrosis, or the peritoneal cavity, a chemical as well as a microscopical examination of it may be necessary before its real nature can be ascertained. Each of these fluids possesses well-marked distinguishing characteristics. For example, the fluid from a hydatid is a pale, slightly opalescent, limpid, alkaline liquid, of a specific gravity of between 1007 and 1014, loaded with chloride of sodium, but containing neither urea

nor albumen—at least the presence of albumen is the exception, not the rule. When examined microscopically it is found to contain, though not always, hooklets and shreds of echinococci, together with plates of cholesterin, as represented in fig. 28. Besides these elements there are sometimes, as Dr. Bristowe found, a number of deeply red-coloured rhomboidal plates of cholesterin floating among the colourless ones. Some of the cholesterin crystals

Fig. 29.



Hæmatoidin Crystals.

were, in Dr. Bristowe's case, studded over with ruby-red hæmatoidin crystals. The case was that of a middle-aged man who died of phthisis (Path. Trans. vol. iv. p. 166). Free hæmatoidin crystals are by no means uncommon products of hydatid fluid, even when there exists no other evidence of blood extravasation having occurred. They are identical with those found in old blood clots, though generally bigger (fig. 29).

Ovarian fluid, being albuminous, is consequently, like ascitic fluid, coagulable by boiling. It contains no urea, nor any great amount of chlorides, thus differing from urine and hydatid fluid. Ovarian fluid is always of a specific gravity of more than 1011. It is viscid and glutinous to the finger touch. It may even be grumous. It sometimes contains cholesterin crystals, but never hooklets.

Ascitic fluid is of a pale amber colour, or, if tinged with bile, of a saffron hue. It is highly albuminous, and has a specific gravity of over 1012. It contains neither urea nor any excess of chlorides.

Hydronephrosis fluid is merely concentrated urine, and consequently gives all the ordinary reactions of urine in a marked form. Sometimes it is albuminous.

If the distended gall-bladder has been punctured, then it is either bile or white mucous secretion which comes from it.

If the tumour be an ordinary abscess or a suppurating hydatid, pus is the fluid obtained.

It must not be forgotten that any one of the six fluids brought away during the exploration may chance to be puriform, in consequence of suppuration accidentally existing in the sac from which it came. But *pure* pus only comes from an abscess; pure bile from a gall-bladder; pure viscid and albuminous fluid from an ovarian tumour; limpid non-

albuminous fluid from a hydatid ; clear albuminous liquid from ascites ; urea in hydronephrosis ; chlorides in hydatid as well as in hydronephrosis ; cholesterin both in hydatid and ovarian, and hooklets only in hydatid liquids.

Finally, let it be borne in mind that the fluid coming directly from a true hepatic hydatid—living or dead—does not necessarily contain either hooklets or shreds of the entozoon. So that one must be on his guard not to be led into an error in diagnosis from not finding morphological evidence of an entozoon in the withdrawn fluid.

In 1868 I read a report of a case of this kind by Mr. Fearn which is worthy of quotation. 'A man was admitted into the Derby Infirmary for a tumour connected with the liver, and on December 8 Mr. Fearn passed a small trocar into it and evacuated eighty-five ounces of fluid, of the appearance of pure water. Its specific gravity was 1012 ; it contained no albumen, no sugar ; and nothing was detected in it under the microscope. On the 24th of the same month the patient was discharged, greatly relieved. In the following March forty ounces of turbid whey-like fluid were evacuated, and the cyst then injected with diluted tincture of iodine, which caused some pain and smarting. Altogether, one hundred and twenty-five ounces of fluid were removed, of which the first eighty-five were as limpid as water,

and contained no echinococci. After fifteen months the patient had perfectly recovered.'

Supposing no fluid, except a few drops of blood, comes away with the exploring needle, all that the operator has to do is to twirl about the end of the needle in the tumour in order to detach a fragment of it, and then slowly withdraw it. Should an aspirator have been employed, if suction be made at the same time that the point is being twirled about, plenty of material will be obtained for microscopic purposes. When placed upon the slide, should the fragment chance to be encephaloid, the kind of cells depicted in fig. 25 will be found. Whereas if the tumour happen to be a syphilitic deposit the material brought away will be such as was described at page 932.

Hydatid associated with other Forms of Diseased Liver.

It must not be imagined that when once a hydatid of the liver has been correctly diagnosed, there is no further need of careful clinical examination; quite the contrary, for the benign entozoal disease may be associated with another, or even others, not of a benign character. A striking example of the truth of this assertion is afforded by a case brought before the Pathological Society, in 1873, by Dr. Silver, where a man had at one and the same time an hepatic hydatid, a cirrhosed liver, and a cancer of the stomach. The patient, aged 49, came into

Charing Cross Hospital with ascites, and a hard mass on the left side of the abdomen. He was tapped, but died from exhaustion. At the *post-mortem*, there was found a large hydatid cyst in the right lobe of the liver; the left was cirrhotic; while the posterior wall of the stomach and the glands behind it were one mass of cancer.

Hepatic Hydatids implicating Blood-vessels.

Having already referred to a case of almost complete obliteration of the vena cava by a cancerous tumour of the liver, I have now to mention that Dr. Elias reports a still more curious case, where not only was the vena cava, but also the portal vein, obliterated by an enormous hepatic hydatid in a patient aged 42, in whom there were great ascites (nearly a gallon of fluid having been removed from the abdomen by tapping) and marked dilatation of the superficial veins of the trunk. The patient was slightly jaundiced; his bowels were constipated. He had dyspnœa and pain in the abdomen. After the withdrawal of the ascitic fluid, the hydatid tumour of the liver became perceptible, but unfortunately the patient died eighteen hours after the paracentesis. At the necropsy, the liver was found to extend up to the third rib, and down to the ilium, projecting forwards, and filling up both sides of the abdomen; the lower portion of the liver

being occupied by a colossal hydatid, divided into two halves, each of which contained a separate mother hydatid, in whose fluid floated numerous daughter vesicles. There was jaundice, and the gall-bladder was somewhat distended, although its ducts were all pervious. Therefore the flow of bile must have been interrupted through the ducts by simple pressure. The main trunk of the mesenteric vein was impervious, and its coats thickened, while the portal vein, for two and a half inches, was a mere flattened cord, scarcely distinguishable from the tissue of the hydatid cyst. The vena cava, from the diaphragm down to the iliac veins, was perfectly obliterated; so the blood had found its way upwards through the anastomosed abdominal and thoracic veins.

A noteworthy case of an enormous hepatic hydatid associated with hæmorrhage, occurring in a gentleman aged 29, and from which was withdrawn by tapping nineteen and a half pints (390 ounces) of clear, limpid, colourless hydatid fluid of a specific gravity of 1007, is recorded by Dr. John Harley in the forty-ninth volume of the 'Medico-Chirurgical 'Transactions.'

'The patient detected the swelling four years previously. It slowly increased, without interfering with any function whatever. The tumour reached downwards till within two fingers' breadth of the

pubes, and as high up as the nipple. Neither liver nor spleen could be felt. In this case the last pint of the fluid evacuated was grumous, and of a bright yellow colour, from the admixture of bile. A number of hydatid vesicles the size of peas, and gelatinous fragments of hydatids the size of walnuts, were discharged along with the fluid. The canula was retained in the cyst until the eighth day, when it slipped from the wound. On replacing the canula twelve hours later, about eight ounces of yellow, turbid, offensive-smelling fluid immediately came away. On the fifteenth day the canula became stopped up with hydatids, and on an elastic catheter being introduced through it it entered to the depth of nine inches, and on its withdrawal twenty ounces of yellow, slightly viscid fluid, containing broken-down hydatid walls, were with some difficulty evacuated.

'It was calculated that up till this time about twenty ounces of fluid had drained away every twenty-four hours. On the forty-third day the canula was replaced by an elastic catheter. On the fiftieth day twenty ounces of fœtid fluid were withdrawn, and from this time the feverish symptoms, from which the patient had suffered, subsided. But the day afterwards dangerous hæmorrhage into the sac took place, and his pulse rose to 160. Vomiting occurred. The skin became hot, dry, and jaundiced.

The epigastric and hypochondriac regions were occupied by the hard distended sac, but the rest of the abdomen was soft and natural. A pint of thick, fœtid, bloody fluid, of the same rich chocolate-red colour as the blood which exudes from the cut surface of a fresh liver, was removed from the sac. Fragments of cyst-wall continually obstructed the eye of the catheter.

'For many days the fæces were completely destitute of bile, of a glistening aspect, and putty colour. One evening the patient passed a very copious, pultaceous, shiny stool, having the same rich reddish-brown colour as the discharge from the sac; from which it appeared that the sac had relieved itself of a portion of its contents through the intestinal canal.

'During the next week much disintegrated blood-clot came away, and oozing of blood within the sac continued. . . . The jaundice disappeared on the fifty-third day. . . . The alvine secretions, however, continued to be entirely destitute of bile, and were very offensive. The discharge from the sac had contained a large quantity of bile from the first, and the proportion of this secretion appeared now to be on the increase. After the sac was washed out, a stream of pure bile flowed from the orifice of the catheter. The discharge of bile continued from the fifty-seventh till the sixty-fifth day. On the eightieth day the fæces became natural. On the hundred and

forty-eighth day, that is to say, rather more than five months after the tapping, the patient resumed his ordinary occupation.'

Throughout the whole treatment it was found necessary to support the lower part of the chest and abdomen with laced bandages. The absence of bile from the intestines was supplied by giving him nightly, with occasional intervals, twenty grains of inspissated ox-bile. For its absence from the intestinal canal was 'always associated with a disgust for fat of all kinds, and the putty-coloured fæces contained undigested, unemulsified oil, which gave to them a soft, glistening appearance like that of frosted silver.'—(N.B. From these last-named facts it is evident that the pancreatic as well as the bile duct was occluded. See page 771 in the chapter on the chemistry of the excretions.—G. H.)

Suppurating Hydatids and their Modes of Termination.

Suppurating hydatids, like ordinary abscesses of the liver, may burst spontaneously in any direction. They may open and discharge their contents into the pleural cavity, the lungs, the stomach, the intestines, the pelvis of the kidney, or into the peritoneum, and by so doing give rise to grave complications, as well as to errors in diagnosis, from the organ that has become secondarily affected being itself diagnosed as the primary seat of the disease. I shall give some

citations which will act as caution-signals in diagnosing these cases. The first one I select is that of a girl, aged 17, who died in the Middlesex Hospital, under the care of Dr. Thompson, from the bursting of a suppurating liver hydatid into the right pleura. She had been in what was supposed to be perfect health up till a fortnight before admission, when she was suddenly seized with acute pains in the region of the diaphragm and right side of the chest, greatly aggravated on deep inspiration. The pain soon became accompanied with dyspnœa, cough, and febrile symptoms, the pulse rising to 112. The whole right side of the chest being at the same time dull on percussion, and no respiratory murmur audible. Hectic set in, and she died one month from the commencement of the attack. There never was any jaundice.

On *post-mortem* examination the right pleura was found full of pus, in which floated hydatid vesicles, from the size of a pin's head to that of a man's fist. From the back of the right lobe of the liver projected a cyst as big as a child's head. It was firmly adherent to the diaphragm, through a hole in which, measuring one and a half inches in diameter, it had discharged its contents. The interior of this cyst was full of pus and hydatids. So that the case was simply one of suppurating hydatid.

A more difficult case than this to diagnose

correctly it is scarcely possible to imagine; for although the liver was the organ primarily affected, not a single symptom of morbid action did it present. While the right lung and pleura—neither of which primarily took an active part in the disease—had all the appearances of being solely at fault. The only symptoms being those of pleuritis with effusion, notwithstanding which neither of these conditions existed.

I will now refer to a case of suppurating hydatid which proved fatal by bursting both into the lung and peritoneal cavity. The case was that of a man aged 19, in King's College Hospital, under the care of Dr. Beale. At the time of his admission he looked anæmic and distressed. Had dull pain and great tenderness to touch on right side, with dulness at base of lung, and inaudible respiratory murmur behind. With a to-and-fro rubbing sound one inch below, and a little to the outer side of, the right nipple. The abdomen measured one inch more on the right than on the left side, with a perceptible bulging. Had constant cough, and three weeks after admission expectorated about four pints of fœtid brownish-green purulent matter in the space of half an hour. When the lungs were afterwards examined, large crepitation, amounting almost to gurgling, was heard all over the right lung base. Two days later he expectorated another pint and a half in two hours.

After which the bulging of the side disappeared. He gradually improved and was discharged as convalescent, exactly three months after his admission into hospital. In six weeks, however, he returned as bad as ever. Three weeks later he was suddenly seized with shivering, and severe pain in the lower part of the abdomen. Nausea and dyspnœa followed, and he died on the following day in a state of complete collapse.

At the *post-mortem*, in the abdomen was a quantity of pus, and the intestines were matted together with effused lymph. The front part of the liver (which was very large) was coated with lymph, and the right lobe adhered to the abdominal walls. Between the gall-bladder (which was shrunken and contracted round some gall-stones) and the liver itself was an abscess the size of an orange. The lower lobe of the right lung and the upper surface of the right lobe of the liver were intimately adherent, and an opening large enough to admit the point of the finger formed the communication between them. While in the lung itself was a similar cavity to one in the liver, also containing the remains of hydatids. The immediate cause of death was peritonitis from the bursting of the hydatid into the abdomen. For further particulars of this interesting case, see Dr. Pollock's report in the Pathological Society's 'Transactions,' vol. xvi. p. 155.

Hepatic Hydatids bursting into the Digestive Canal.

Hepatic hydatids occasionally, ay, frequently, burst directly into the intestines, and that is the most favourable way in which they can spontaneously empty themselves.

The following very good example of a case of hydatids of the liver passing away by the intestines is related by Dr. Sinclair Holden in the 'British Medical Journal' of July 10, 1869.

A woman, aged 60, enjoyed good health till nine months before her decease, when the liver became slightly enlarged. A month before her death she passed by stool portions of a cyst, having attached to its inner surface a number of pedunculated sacs of the size of a pea. The microscope showed hooklets.

M. Arles (Montpellier) communicated the case of an elderly woman who had a voluminous tumour in the right hypochondriac region. After exhibiting symptoms of slight peritonitis, she passed *per anum* an hydatid cyst. It contained neither hooks nor any membranes, only a small quantity of fluid not unlike bile. The microscope revealed the existence of an acephalocyst.

A much more curious case than either of these came under my own notice several years ago. The patient, a middle-aged gentleman, who had been suffering from slight jaundice for a considerable time,

was sent to consult me in 1871 by Dr. Henry Nuttall, of Leicester. The case was one of hydatid cyst pressing upon and partially occluding the common bile-duct. There was nothing unusual or remarkable about the case, except that after a time the patient began to pass hydatids along with the stools, and continued to do so at intervals during the whole first six months of 1872—in fact more or less up till the time of his death, which occurred in the month of August in that year. How many hydatids he actually passed it is impossible to say, but the following extracts from two of Dr. Nuttall's letters, respectively dated April 29 and June 7, 1872, furnish ample evidence of their number having been very great. As these letters were not written with the idea of publication (for at the time neither Dr. Nuttall nor I dreamed that the case would ever appear in print), they are not to be regarded as clinical reports, but merely as ordinary communications, the value of which consists in their being thoroughly impartial statements of bare facts.

'April 29, 1872.—Mr. P., who has consulted you on different occasions, has for some few weeks been passing hydatid cysts *per rectum*, and is anxious for you to express some opinion, based upon your knowledge of his past state, as to his probable future, now that cysts are undoubtedly passing from the liver

through some abnormally formed channel into the intestinal canal.

'His general state of health is much impaired, but he does not appear to be getting worse—indeed he is constantly saying how much better he is. There is a good deal of emaciation, but not apparently increasing. His skin is remarkably yellow, and the infra-orbital parts of the face very dark. Appetite good. Motions of a pale colour. Water contains a good deal of bile as well as some lithates.

'I have been hoping, now that a fairly free communication has established itself from the cyst to the intestines, the patient would improve a little more; but the abdomen continues hard, and from its irregular lobulated surface, I fear there may be more cysts than one, in which case it requires a sanguine disposition to trust to opening after opening forming until there be a channel of outlet for the whole.

'Mr. P.'s undaunted spirit admits of no doubt; but we may well wonder how ever the wonder-working powers of struggling recovery will bring about the desired end.

'We are not justified, I think, in proposing any surgical external opening so long as cysts broken and entire continue to pass through the bowels.

'June 7.—Mr. P. called upon me to-day after an absence of perhaps twelve or fourteen days, and I find a good deal of œdema of the lower extremities.

The tumour in the abdomen is still hard and large, and the colour of the countenance remarkably dark, as much so as if nitrate of silver had been given for a very long time. Although hydatids continue to pass through the bowels, I fear there is not much substantial progress. It seems to me that the discharge of the hydatids does not take place from the entire mass of the tumour, probably in consequence of its being composed of more than one cyst. The appetite is good; rest fairly so; bowels costive and requiring daily enemata; urine fairly copious and clear; no albumen.' The patient died on August 10. He had been gradually getting weaker and weaker, and a week before his death sickness and exhaustion set in.

Hydatids may burst into the Lungs without proving Fatal.

In order to prevent what might otherwise be an embarrassing error in prognosis, it is necessary for me to call special attention to the fact that even the bursting of a suppurating liver hydatid into the lung is, contrary to what might be expected, by no means a necessarily hopeless result. For it has happened that not only the purulent contents of the cyst, but hydatids themselves, have found their way into the bronchi and been safely expectorated. This kind of expectorative evacuation has been known to continue for days, until the hydatid cyst, indeed, has not only completely emptied itself, but become

shrivelled up and closed, and the patient got well and lived for months afterwards. Even hydatid vesicles as large as chestnuts have been expectorated.

In every case where the presence of hydatid vesicles is suspected in sputa, the patient should be made to expectorate into a tumbler of pure water, when, if any vesicles are present, they will be seen floating about in the water.

Being thus aware of the chances of the patient's recovery after the bursting of a parent hepatic hydatid into the lungs, whenever a case of the kind occurs strict rest is to be enjoined, and all possible soothing measures adopted, in order, as far as possible, to moderate the rapidity of the discharge of the daughter-vesicles, so as to lessen the risk of sudden suffocation from the bronchi getting blocked up. If this is successfully accomplished, the patient is almost sure to recover. There are even cases on record where hydatids have emptied themselves by the lungs in the short space of a few days, collapsed, and completely healed up.

An Hepatic Hydatid may prove Fatal by inducing Secondary Pulmonary Disease.

That a suppurating liver hydatid, by inducing secondary pulmonary disease, may prove fatal without bursting either into the lungs or pleuræ, is shown by the case reported in the 'Lancet' of May 8, 1869,

which occurred in St. Mary's Hospital under Dr. Sieveking.

A lad, aged 17, was admitted suffering from pleurisy with effusion. About a month before he felt a pain in his right side. This pain was worse when he lay down. He soon experienced difficulty in breathing in going upstairs, and was obliged to leave off work.

Breathing was good over the upper half of right side; below, was absolute silence and dulness. Liver pushed down lower than hypochondrium. A few days afterwards the edge of the liver was felt a quarter of an inch above the umbilicus; the veins on the surface of the abdomen much distended; pain and tenderness all over abdomen; fluctuation at a point about two inches above and to the right of umbilicus. A day or two later, the liver was punctured by a trocar, and about eight ounces of fluid, with small gelatinous masses, was drawn off. The canula was plugged and left in. On the following day, two or three ounces of a similar fluid was evacuated. Had a rather restless night; pulse 128, small and wiry. Patient becoming emaciated; pain in the abdomen much relieved since operation. Two days later, an incision was made, and about ten ounces of offensive pus, with a large quantity of gelatinous masses containing hydatids, evacuated. On the next day, fifty ounces of extremely offensive pus was discharged. The

liver was thus reduced in size by about two inches, and tension removed. No tenderness, except at the edge of the wound. The patient died the next day, and at the *post-mortem* the liver occupied nearly the whole front of the abdomen, and was generally adherent to the front abdominal wall (the spot of puncture being firmly adherent). Left lobe very much enlarged. Right lobe almost entirely converted into an abscess containing pus, with flakes of lymph and gelatinous cysts.

Sudden Death may occur from Hydatids.

A jaundiced woman, æt. 29, in 1855, died in twelve hours in University College Hospital from hæmorrhage following severe spasmodic epigastric pain accompanied with a discharge of blood from mouth and anus. At the *post-mortem*, Dr. Hillier—who reports the case in the seventh volume of the Pathological Society's 'Transactions,' page 222—says that the liver with its contents weighed ninety-one ounces; when emptied of them, fifty-four ounces. The right lobe contained a large hydatid, the interior of which was full of blood. An aperture led from the wall of the hydatid into the bile-duct, through which the blood had flowed into the intestines—for the duodenum was full of fluid blood. The hæmorrhage was traced to a perforated hepatic artery.

As an example of sudden death following upon

the accidental bursting of a non-suppurating hydatid into the lungs, I may cite a case which occurred in the Cheltenham Hospital, which was recorded by Mr. Carden. It was that of a girl, six years old, suffering from what was supposed to be tubercular phthisis. She had been ill three years, and on admission her breathing was oppressed, difficult, and short, and she had a peculiar hollow cough. The child sat up in bed, had a coughing fit, fell back and expired. *Post-mortem* examination: The right pleura was adherent, the left one partially so, with no fluid in either one. The lower lobe of the right lung was one large fluctuating tumour, with a little healthy lung at the base. On cutting into the tumour, about a pint of perfectly clear watery fluid escaped. The cyst wall was a pearly-white membrane, one-sixteenth of an inch thick, and the whole could be lifted out entire, not being adherent to the cavity which it had formed in the lung. Another large cyst, having the same characteristics, occupied the whole of the upper lobe of the left lung, there being only a little fringe of healthy lung round it. In the longitudinal fissure of the liver another cyst was found, containing about six ounces of fluid. This one, however, contained about a dozen secondary cysts, about a quarter of an inch in diameter, and floated on the surface of the fluid. The fluid was slightly alkaline, containing a trace of albumen; the

specific gravity being 1010. The deposit from the fluid revealed hooklets, and small but perfect specimens of the *Echinococcus hominis*.

Even the bursting of the cyst into the peritoneal cavity is, however, far from being free of danger, as in the majority of instances the presence of hydatid fluid and hydatid vesicles in the peritoneum sets up a rapidly fatal form of peritonitis, as exemplified in a case already cited at page 978, and also in the case reported at page 1008.

Spontaneous Cure of Hydatids.

Liver hydatids oftentimes of themselves undergo a spontaneous cure by the parasite dying, the fluid being absorbed, and the tumour collapsing, and either becoming calcified, shrivelled up, or ending in becoming a cheesy or putty-like mass,[1] whose true origin is only discoverable by the finding within its substance hooklets of echinococci when it is carefully examined with the microscope.

Sometimes again a spontaneous cure takes place by an idiopathic inflammatory action being set up in the hepatic hydatid, and adhesion taking place be-

[1] A man of 36 years of age was admitted under Dr. Murchison's care into the Fever Hospital, in the right lobe of whose liver was found, at the autopsy, a hydatid which, curiously enough, was so full of secondary cysts as to contain no fluid, but only a putty-like material. While the outer parent cyst-wall had the appearance, in some places, of commencing calcification. Just as if a spontaneous cure was contemplated by means of consolidation. The case is reported in the *Pathological Society's Transactions*, vol. xviii. pp. 123–5.

tween its walls and the neighbouring tissues—the gall-bladder, the bile-ducts, abdominal parietes, the diaphragm, the pleural cavity, the stomach, the intestines, or the urinary bladder. The tumour then ends by the bursting of its cyst, and the discharging of its contents to the exterior of the body, through the intermediary of the organ to the parietes of which it has attached itself by the adhesive inflammation. The bursting of the cyst in this way is unattended with any danger, except when the cyst has attached itself to a blood-vessel—such as the vena cava for example—or indirectly to the lung through the medium of the diaphragm, and discharges itself in so great quantity and so rapidly into the bronchial tubes as to cause suffocation or set up acute and fatal inflammatory action.

Treatment of Hepatic Hydatids.

During the last half-century a great number of remedies have been proposed, used, and gradually abandoned as totally inert in the treatment of hydatid disease of the liver. Every possible or impossible anthelmintic has had its trial, and all have in turn succumbed to the stern edict of experience—which has invariably delivered its verdict in the condemnatory though pithy word 'useless.' Even within the last few years several new hepatic hydatid helminthicides have been proposed. The iodide of potassium, for example,

was proclaimed by some of our Continental brethren as a specific, and was for a time much employed, under the impression that, like the oil of male fern, it had the power of killing the entozoon. I too gave it a trial, but never in one single instance did I find any good effects follow its employment, and I doubt its efficacy, seeing that on one occasion I actually saturated a woman by giving her twenty grains of the drug thrice a day for six days running, until both her saliva and her urine were loaded with it, as was seen when the usual starch test was employed. Yet curiously enough, in spite of this blood saturation, the same reagent actually yielded no result whatever when it was applied to the fluid drawn off by a trocar from the hydatid cyst itself. Which, by its projecting immediately beneath the edge of the false ribs a little to the right of the nipple line, was easily got at. The fact of the hydatid fluid being found to contain no trace of the iodide, when the blood was known to be thoroughly saturated with it, I consider as being sufficient of itself to prove not only the existence of a non-affinity between the iodide of potassium and the hepatic hydatid secretion, but that no specific action can consequently be exerted by the drug on the parasite which it thus seems to avoid.

Not only do I regard iodide of potassium as useless ; but I believe that not one single drug in the Pharmacopœia possesses the power, when administered

by the mouth to the patient, of poisoning or otherwise destroying the parasite, and, so long as it lives, a cure of the disease is hopeless.

In my opinion, there is at present but one known specific cure for hydatid disease of the liver, and it may be summed up in the simple word 'evacuation,' which means the evacuation of the fluid of the hydatid by means of a fine trocar, or better still by an exploring needle, which simple operation is not only free from danger, but in most cases sufficient to effect a speedy cure. As a striking example of the truth of this remark, I quote the following case. In 1874, Dr. Macaldin, of Tavistock Square, consulted me regarding the case of a girl of 12 years of age who had a large and exceedingly hard stone-like swelling or tumour, extending from the right side of the liver down into the iliac fossa, and latterly to an inch or so beyond the left of the umbilicus. It had existed for twelve months. Although the tumour was as hard as a cannon-ball, its history and position led me at once to diagnose it as a case of hepatic hydatid. Acting on this opinion, with which Dr. Macaldin unhesitatingly agreed, I thrust an exploring needle into the tumour as the girl lay on the sofa of my study, without making any preparation whatever, but holding a jug in readiness. Immediately on the instrument being inserted, there spurted out a clear opalescent liquid, which, on being chemically tested,

at once revealed its nature to be that of hydatid fluid. While I applied the necessary tests to the first portion of the liquid which came away, Dr. Macaldin attended to the patient, and as, when about a pint of liquid had drained off, it began to come away only in driblets, at my suggestion he pressed upon the tumour firmly with his right hand, while in the left he held the jug to receive the liquid discharged from it until fluid entirely ceased to escape. By which time the tumour had completely collapsed. So the canula was withdrawn, and nothing more than a piece of sticking plaster put over the opening. The girl then rose from the sofa as if nothing whatever had been done to her, and left my study, to return home two days afterwards hale and hearty, along with her mother, by train to the midland counties. In 1879, that is to say four years afterwards, Dr. Macaldin told me that he had heard from the relations (her mother died of cancer of the uterus twelve months after she brought her daughter to London) that the girl rapidly regained health and strength after her return home, and that she not only never had a return of the disease, but was then, at the age of sixteen, a robust young woman. The fuss that is made about tapping hydatids, once they have been correctly diagnosed, is, according to my experience, quite uncalled for, as I have never seen a bad symptom follow the operation when properly performed. There

is, however, one source of danger which I must call attention to, for it is not even so much as alluded to in any work on the subject I have ever read. It is the danger that is occasionally run, during the simple operation of tapping, from the sudden fatal effects of air entering into an accidentally transfixed blood-vessel, in spite of the fact that the entrance of air into the cyst itself is of no importance whatever.

That the operation occasionally leads to a fatal result, in consequence of air finding its way into a blood-vessel accidentally transfixed during the operation, is illustrated by the case reported by Mr. Bryant in the eleventh volume of the Clinical Society's 'Transactions.' The case is that of a man aged 40, who suddenly expired after having been tapped with a fine trocar $3\frac{1}{2}$ inches to the right of the median line, and immediately beneath the edge of the ribs. The instrument pierced through about an inch of the liver, and drew away nine ounces of clear watery non-albuminous fluid. A pad of lint was immediately applied to the opening, but in a few seconds the patient became intensely flushed, and complained of agonising pain *in his face and jaws*. He next became livid, then unconscious, vomited once or twice, had a sort of fit, and expired. At the *post-mortem* it was found that the trocar had transfixed the trunk of the portal vein, just before dividing into its two largest branches, and Mr. Bryant says that

he is of opinion that after the withdrawal of the canula hydatid fluid entered the vein, and acted as a fatal poison.

I object to this theory on the following grounds. Firstly: The symptoms described are assuredly not those of a toxic agent introduced into the circulation. Secondly: It is highly improbable that sufficient hydatid fluid could enter the vein rapidly enough to produce so suddenly fatal a result. Thirdly: The entrance of air into the portal vein, by being sucked as it were into it through the canula, just at the moment its distal end was being withdrawn through the centre of the transfixed vein, is an exceedingly likely thing to have occurred. While, lastly, all the symptoms manifested by the patient so closely resemble those known to arise from the accidental admission of air into the circulation, as to leave no doubt on my mind that it was the admission of air into the circulation, and the admission of air alone, that killed the patient.

The admission of air into a vein and the admission of air into a hydatid cyst, be it remembered, are two very different things. A cyst, and even a large cyst, may get filled with air, and not a single untoward symptom result from it. Whereas the admission of merely a few air-globules into the venous circulation, especially in close proximity to the heart, is instantly fatal.

How to tap an Hepatic Hydatid.

In performing the operation of tapping, especially if the tumour be a large one, the following points require to be specially attended to :—Select a fine trocar. One not much thicker than an ordinary stocking knitting-needle. Dip it into carbolised oil, and puncture the tumour at its most prominent part. As soon as the trocar is withdrawn, pass a long probe through the canula, and measure the size of the cyst.[1] When fluid has ceased to flow, before removing the canula make sure, by again passing in the probe, that it has not merely got accidentally occluded by a vesicle or débris. When completely emptied, measure the dimensions of the cyst a second time, in order to see how much it has contracted. Immediately after the completion of the operation and the removal of the canula, the opening is to be covered with a piece of sticking plaster, or lint steeped in collodion, over which a compress and bandage are applied. If the hydatid has been large, the patient is to be kept for forty-eight hours in the recumbent posture, and every movement of the body strictly prohibited.

If a small-sized canula fails to admit of the hydatid emptying itself in consequence of its containing

[1] Before the cyst has had time to begin to contract, a probe may penetrate to the depth of eight, ten, or even twelve inches. In the latter case the cyst will hold a gallon or more of hydatid fluid.

solid contents, then one of the size of a No. 12 catheter is to be substituted. A bigger one is scarcely ever necessary. For in by far the majority of instances the No. 12 size is found sufficiently large to admit of the passage through it of daughter cysts, as well as granddaughter vesicles, shreds, and other débris from the walls of the parent hydatid. Should one single operation appear to be insufficient for the complete evacuation of the cyst, then a permanent opening may be established by the simple retention of the canula in the wound. After a time the canula becomes loose, and if necessary it can easily be replaced by a still larger one, until the whole of the contents of the cyst has been evacuated. When it is intended at once to establish a fistulous opening, and treat the case with a drainage-tube, the following points may be considered with advantage.

a. In consequence of every hydatid contracting after the evacuation of its contents in the direction of its base, or point of attachment to the organ from which it grows, when it is intended to insert a drainage tube into the opening the hydatid ought to be punctured at the point supposed to be the one nearest to the seat of its attachment.

b. During the operation no care need be taken to prevent the entrance of air into the cavity of the hydatid ; for after the danger of air entering a blood-vessel during the operation has passed away, there is

no fear of the presence of air in the sac being attended with any deleterious consequences whatever.

c. In order to facilitate the evacuation of hydatid daughter and granddaughter vesicles as well as shreds and débris, a stream of tepid creasotised or carbolised water may be gently, slowly, and steadily passed into the sac by means of an ordinary canula or a double canulated silver male catheter every day, so long as it is deemed necessary.

d. When once adhesion between the opening in the hydatid and the abdominal walls is established, there is no danger whatever from the injection of strange fluids into the cavity—such as solutions of sulphate of zinc or tincture of iodine—if only care be taken to stop the injection the moment the patient complains of experiencing a feeling of heat in the sac.

e. In all cases where bleeding from the walls of the hydatid takes place, solutions of tannic acid, alum, perchloride of iron, and other such like styptics should be injected.

f. In order to favour the collapse of the sac after its contents have been discharged, especially when it is large, a thick compress is to be tightly bandaged over it. This may even be done while a drainage-tube is being kept in the hydatid, by having a hole in the centre of the compress, through which the free end of the drainage-tube or catheter may protrude.

As an illustrative case I may quote one recorded by Dr. Groves ('British Medical Journal,' February 19, 1881), where nine-and-a-half pints, that is to say more than a gallon, of a semi-purulent hydatid fluid, 'containing numerous empty cysts,' were removed by an aspirator from the liver of a woman, aged 35, on June 21. 'The operation occupied an hour. In the course of the following days, there was diarrhœa, and the stools were of pale colour. On July 14, aspiration was again performed. Seven-and-a-half pints of bile-stained fluid were removed. Next day, potassa fusa was applied to the right of the puncture. On the 18th, the cyst was tapped through the eschar. The cavity was washed out with a solution of chloride of zinc. An india-rubber drainage-tube, nine inches long, was introduced through the canula; the latter was then withdrawn, and the tube retained. The cyst was washed out twice daily with a two per cent. carbolic solution. The discharges were deeply stained with bile. For several days the patient became weaker, with sickness and diarrhœa. Bile began to reappear in the stools; and from August 1, there was a great improvement. The stools became natural on August 20, and she continued to improve. In the middle of September the wound was allowed to heal.

The most common cause of danger in the operation, is, I believe, the possibility of inducing suppura-

tion of a healthy hydatid by making too large an opening into it at first. Hence the reason why I am so particular in recommending the employment of a fine trocar, an exploring needle, or the nozzle of a hypodermic syringe. The only disadvantage in the employment of these arises from the longer time it takes to empty the hydatid, and the difficulty daughter vesicles, if there be any, as well as shreds, have in escaping through a small opening. But, seeing that the small canula ensures a minimum of danger along with a modicum of advantage, it is to be preferred, at least in the first instance, and only a large trocar subsequently had recourse to when it is found that there are daughter cysts and shreds which cannot escape through the small canula. My last piece of advice is never to inject either irritant or styptic fluids into hydatids, or even to wash them out with tepid water, unless there be good reason for so doing. Experience having taught me that when healthy hydatid fluid has alone escaped by the trocar, there is fortunately no occasion whatever either to wash out the cyst, or to inject it with an astringent, in order to ensure a successful result.

After the operation of tapping, the extract of male fern, from its well-known anthelmintic properties in cases of tænia, has been injected into the cyst. A case is recorded by Dr. Pavy where a woman aged 21 was thus operated upon. Its history is

briefly as follows. About six years before her admission into Guy's Hospital, she had noticed a swelling in her right side, and at the time of her admission a large tumour caused a considerable bulging of the ribs on the right side, and the right mammary gland was raised about three-quarters of an inch above the level of the left. Fluctuation was apparent. A fine trocar was introduced by Mr. Durham, and about four ounces of a colourless fluid escaped. A liquid consisting of half a drachm (by measure) of extract of male fern, half a drachm of liquor potassæ, and six drachms of water, was then injected into the sac. At the introduction of the trocar the patient complained of considerable pain, which she referred to the lower part of the abdomen. Febrile excitement, vomiting, and purging followed, but there was no evidence of peritonitis. When seen some months afterwards, no swelling was perceptible, but a hardness remained in the hypochondriac region.

The inference drawn by the reporter from the result in this case is, that the injection of the extract of male fern caused an immediate destruction of the life of the hydatid without the production of suppuration, and that a more rapid absorption of the fluid elements of the cyst afterwards took place than would have occurred from simple tapping—a theory regarding the value of which I have grave doubts.

The treatment of hepatic hydatids by electrolysis has been by a few writers recommended in preference to tapping, and a series of successful cases have been recorded by Dr. Hilton Fagge and Mr. Arthur Durham, in which the operation was performed in the manner recommended by Dr. Althaus. From the report of the cases as given in the 'Lancet' of November 19, 1870, I extract the following :—

'The current was allowed to pass for from ten to twenty minutes. The operation was followed in most cases by rapid diminution of the tumour. At the same time, in some cases, fluctuation became perceptible in the lower part of the abdomen. The authors believe that some of the hydatid fluid probably escaped through the punctures made by the needles. The success of the operation would thus appear to depend, not on the direct action of the electric current, but on its effecting, as it were, a kind of subcutaneous tapping; and they suggest that simple acupuncture might possibly be followed by equally successful results. Slight febrile symptoms, and more or less pain, in most cases followed the operation; these symptoms, however, rarely lasted more than three or four days. In one instance they were entirely absent. After the lapse of six months or a year, when each patient returned for examination, no trace of the disease remained; or, at most, there was only some ill-defined fulness of the epigas-

trium. In three cases more than one cyst existed, and each cyst was electrolysed separately.

'Dr. Fagge believes that the process acts by liberating hydrogen, which then forces the hydatid fluid out of the cyst.'

In order, moreover, to show that even the injection of irritating liquids into a hydatid may be attended with advantage rather than serious consequences, I may refer to a case (recorded in the 'Lancet' of February 20, 1875, page 269) where a woman aged 40, who had three hepatic hydatids emptied by an aspirator, and afterwards injected with the tincture of iodine, got perfectly well.

Skoda reported a case of a large hydatid treated successfully by injecting into it a solution of iodine. A man aged 46 suffered from a large fluctuating tumour on the left side of the abdomen, which reached from the upper border of the eighth rib to the crest of the ilium. An exploratory puncture was made, and several ounces of a clear pale yellow fluid drawn off, which contained echinococci. After its removal the following mixture was injected: tincture of iodine, four ounces; water, one ounce; iodide of potassium, one scruple. This injection was allowed to remain for thirteen minutes. In the course of two hours, iodine was discovered in the urine. The patient was subsequently affected with salivation, inflammation of the parotid glands, con-

junctivitis, and swelling of the eyelids. A second injection was made in the following year, which, like the former operation, resulted in severe abdominal pain. The patient gradually improved, and was discharged in perfect health. ('Wiener Med. Zeit.' No. 19, 1868.)

Although these are successful cases of injecting an irritant solution into hydatids, it is by no means to be usually followed. For even the washing one out with a weak solution of carbolic acid has proved fatal. I recollect Dr. Murchison showing me, in 1869, a hydatid taken from the body of a woman who had had a swelling in the hepatic region as long as she remembered, and during three weeks before her death had symptoms of peritonitis and jaundice. The tumour was punctured, and a large quantity of pus and secondary vesicles drawn off. The cyst having been washed out with carbolic acid lotion several times a day, the patient died. At the *post-mortem* a large hydatid was found, containing half a pint of bilious fluid with collapsed cysts.

I have now to call special attention to some peculiar cases which have been wisely put on record by different writers. Some of which are pregnant with clinical materials for thoughtful consideration.

An instructive fatal case of tapping is recorded in the 'Lancet' of January 11, 1868.

A labourer, aged 35, was admitted into the East

Suffolk Hospital under the care of Mr. Bullen on November 2, 1867, with a large fluctuating tumour over the fifth and sixth ribs, and about three inches below the right axilla. The liver was much enlarged. He had a cachectic look. About two days after admission, the swelling, which was intensely painful, began to show symptoms of suppurating, and a small trocar was introduced ; but nothing coming through the canula, a free incision was made, and out gushed a large quantity of fluid and hydatids. Altogether two pints. Some of the hydatids were living ; they varied in size from a pea to a fowl's egg.

On the following morning about a pint came away ; and every succeeding morning, for a month afterwards, more or less escaped.

From the fifth day the fluid became coloured with bile, purulent, and very offensive. Sometimes hundreds of hydatids escaped through the dressings during the night, and the patient became much emaciated, and died on December 6.

On opening the body thirty-two hours after death, the right lobe of the liver was found enormously enlarged and nodulated, adherent to the abdominal parietes and the diaphragm. In breaking down these adhesions, it became torn, discovering an immense cavity in it, full of hydatids, both living and dead, the contents measuring over two pints ; and there were two or three openings from the sac between the fifth

and seventh ribs, through which the hydatids passed during life through the side of the diaphragm and intercostal spaces, then under the integument, and through the two superficial openings of the skin, three inches below the axilla.

Dr. Anstie reported ('Practitioner,' 1872) the case of a girl aged 7, who had a large tumour of the right hypochondriac region bulging from below the ribs, said to have followed a blow. She was so emaciated and cachectic-looking, that cancer was suspected. In fact the case might have been equally well taken for one of the three following diseases : cancer, suppuration after injury, or hydatid. In order to solve the question, Mr. Berkeley Hill explored the tumour and withdrew four ounces of hydatid fluid. Now comes the curious part of the case. The day following the operation a *scarlatinal* rash made its appearance. The fever ran a mild and short course, and the wound healed after having given slight pain for a day or two. This fact of the induction of scarlet fever is exceedingly interesting to the clinical pathologist when considered in connection with what is said by Dieulafoy in his treatise on 'Pneumatic Aspiration.' He mentions four cases in which he had seen a rash, which he describes as 'urticaria with fever,' associated with sore throat and dysphagia, follow tapping of the liver. Dieulafoy's cases from his own description were, I have no doubt, like Anstie's, distinctly scarlatinal. The

rash having covered the body, and been accompanied by distinct evening exacerbations of fever, and associated at the same time with inflamed fauces.

Another anomalous case of tapping, which was followed by exfoliation of the parent cyst and its discharge through the opening about six weeks after the operation, is recorded by Mr. Thomas Bryant in the following words: 'The external opening into the cyst became obstructed, having evidently become plugged with hydatid membrane, a portion of which was projecting externally. An attempt to remove the plug was made, and much of the membrane drawn away; but it proved to be rotten, and the attempt caused so much pain that it was deemed prudent to leave the expulsion to nature's efforts.' The next day 'the whole mass of hydatid membrane was spontaneously expelled, accompanied with a rush of several pints of a dreadfully offensive purulent bile-stained fluid. The swelling of the abdomen at once disappeared, with all pain and constitutional disturbance. The hydatid membrane, which had been discharged, was evidently the wall of the parent cyst. A catheter was subsequently introduced daily through the wound into the cavity, which was carefully washed out,' and in a month 'the wound had completely healed. The discharge had gradually become less day by day, and it soon lost its offensive character.' In a month more 'she left the hospital

with no signs whatever of the abdominal tumour, not even with an induration ; the small cicatrix of the wound, which had been made by the trocar and canula, alone remained.' (Pathological Society's 'Transactions,' vol. xvii. p. 168.)

In a case related in the 'Gazette des Hôpitaux' of August 10, 1875, half a gallon (2,400 grammes) of fluid was withdrawn at the first tapping from a hydatid of the liver of a man aged 25. Six days afterwards it was again tapped ; but this time, instead of colourless non-albuminous hydatid fluid, a pint of bile-stained clear liquid was withdrawn. Within the next twelve weeks he was four times tapped, and a pint, three pints, two and a half pints, and three and three-quarter pints of fluid were successively withdrawn, each tapping yielding more and more turbid fluid, and the last containing coagulable albumen, bile-pigment, and a quantity of leucocytes. An alcoholic injection was now had recourse to ; but it was followed by shiverings, fever, and vomiting, and in ten days later, when the cyst was again tapped, it yielded nearly three pints of purulent fluid. A fistulous opening was next attempted to be established by Vienna paste, but this was speedily followed by peritonitis from which the patient succumbed within eight days. At the necropsy the whole right lobe of the liver was found converted into a large fluctuating sac, which contained nearly five pints of

whitish purulent fluid and weighed altogether nearly ten pounds. A number of daughter vesicles containing clear normal hydatid fluid in their interior floated in the purulent liquid ; so it would seem that the daughter cysts were unaffected by the suppuration in their parent. The death of the patient in this case was attributed to extensive suppuration in the parent cyst associated with an extension of the inflammation to the peritoneum. The intestines having been found glued together by soft false membranes.

As a supplement to hepatic hydatids I may refer to an exceedingly rare case of

Distoma Hepaticum (Liver Fluke) in Man,

published in the 'British Medical Journal' by Drs. Humble and Lush. The history they have given of the case is the following :—

A labourer, aged 52, complaining of vomiting, with pain at the upper part of the abdomen, was admitted into the Dorset County Hospital on November 21, 1879.

His abdomen was distended with flatus, intensely tender. His pulse was 72, temperature 98°. There was no vomiting, but he stated that he had been suffering from vomiting for two months to such a degree that he had been unable to retain any food. Four days later there were increased pain and tenderness, pulse 102, temperature 101·3°. No increase of

hepatic dulness could be detected. On November 28, evacuations were passed involuntarily on coughing. The breath was very offensive. On December 6, he vomited half a pint of coffee-ground like fluid, with a dark greenish scum. Delirium continued more or less for several days, and subsided about the 11th. The next day, a large ash-coloured stinking slough, about five inches by three, was discharged *per rectum*. Though quiet and sensible, his motions were passed involuntarily. So he continued for some weeks. At length, fistulous abscesses formed in the neighbourhood of the rectum. He died March 31, 1880.

At the necropsy, the upper part of the rectum was found thickened and narrow, and fistulous abscesses opened into it. The liver was of a greyish red, and easily broken down. The hepatic ducts were enlarged, and contained twenty-six distomata hepatica.

Remarks.—His widow states that he rarely ate animal food, never sheep's liver nor rabbit. He occasionally ate calves' lungs, but not the liver; his diet was chiefly bread and cheese; he frequently partook of watercresses.

CHAPTER XXI.

CYSTIC DISEASE OF THE LIVER.

Contrary to what might be expected from the frequency with which one meets with cases published under the title of 'Cystic Disease of the Liver,' this is an extremely rare form of affection. The vast majority, I might say perhaps 90 per cent., of the tumours bearing the title having no right whatever to be so named, except in so far as they are represented as being more or less globular and possessing encysted contents. From the word 'cyst' being simply the anglicised Greek for bladder, it may of course be legitimately applied to every form of bag-like growth with all kinds of fluid or solid contents. But as the employing it as a noun is an entirely different thing from the using it as a distinguishing adjective applied to a special form of disease, I beg to call attention to the following particulars.

'Cystic disease of the liver' is the exact analogue of cystic disease of the testicles, ovaries, or kidneys, and from being, in the majority of instances, due to congenital constitutional causes, is frequently

associated with them. Consequently it ought not to be confounded with merely acquired encapsulated diseases, such as hydatids, cancerous nodules, purulent deposits, the ends of dilated bile-ducts, &c. All of which have of course a perfect right to be spoken of as cystic, if their defining title be associated therewith ; as in the terms hydatid cyst, purulent cyst, &c. The term ' Cystic Disease of the Liver ' is, however, quite another thing, and is a name *sui generis*, which ought not, for example, to be applied to cases like those described at pages 115 and 961, or the one to which Abercrombie refers, where the distension of the ordinary hepatic ducts within the liver substance with pent-up bile was so great as to give to the parenchyma of the organ the appearance of being studded over with cysts. The greater part of the secreting tissue having become so atrophied and wasted away, from the backward pressure exerted upon it by the pent-up bile, that the liver looked as if it were a great sac. The cause of all this being a membranous obstruction in the common bile-duct.

True cystic disease of the liver is, I believe, like cystic disease of the kidneys, due to congenital structural tissue malformation, as I explained in a case of multiple cysts which were found in the right kidney of a child aged 10 months (Pathological Society's ' Transactions,' vol. xv. p. 146). So it is not surprising to find that true cystic disease of the liver

may be associated in the same patient with cystic disease of the kidney.

Both Drs. Bristowe and Wilks, who have given special attention to the subject, have reported several cases of this association in various volumes of the Pathological Society's 'Transactions.' At page 175 of vol. x. Dr. Bristowe describes the cyst walls as being composed of fibrous tissue, similar to that of a serous membrane, and their general contents as being a serous fluid. In fact their pathology is identically the same as that of the true cystic diseases of the kidney, choroid plexus, and all other organs of the body. Dr. Bristowe met with a case in 1859 in a woman whose liver, though of normal size, contained a vast number of small cysts filled with serous fluid of a reddish-brown colour. The kidneys weighed (together) $17\frac{3}{4}$ ounces, and were also thickly studded with similar cysts, the largest of which was of about the size of a pigeon's egg.

Dr. Pye Smith exhibited to the Pathological Society (vol. for 1881, p. 113) specimens of cystic disease of both the kidneys and liver taken from a man aged 50, a drunkard. The kidneys together weighed sixty-four ounces. The liver contained one large and several smaller cysts.

In January 1882, Dr. Sharkey showed to the Pathological Society specimens of cystic disease of the liver from a female, aged 38, who died from frac-

ture of the skull. There were cysts in the ovaries also. The liver substance was pale and firm. The cyst in it was round, with thin walls, in which vessels could be seen. Septal cords and membranes were found traversing its cavity. The fluid was alkaline and of a straw colour; contained much albumen, and chlorides. The cyst-wall was fibrous and lined by a single layer of flat epithelium-cells. Mr. Doran said he thought that there was an homology between cysts immediately under the capsule of the liver and the cysts found around the Fallopian tubes. But these latter cysts are not in the substance of the wall of the tube, but developed in the folds of the broad ligament.

Although it is most commonly hydatids and dilated bile-ducts which are erroneously spoken of as cysts, even cases of cancer have been so described. For example, a well-marked case of this kind Dr. J. W. Ogle brought before the Pathological Society in 1858. The specimen contained a cavity as large as a cricket-ball filled with a light-coloured fluid with broken-down carcinomatous matter floating in it. Its walls consisted of a $\frac{3}{4}$-inch thick layer of soft yellowish-white carcinomatous deposit, which deposit was itself separated from the healthy hepatic tissue by a capsule-like investment. But of course this was not a case of 'cystic disease.'

CHAPTER XXII.

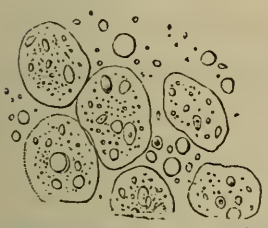
BENIGN DEGENERATIONS OF HEPATIC PARENCHYMA.

Fatty Liver: its Etiology and Pathology.

Fatty disease of the liver is exceedingly common in this country, not alone among human beings, but amongst all overfed domestic animals, more particularly of the porcine, ovine, and bovine species. Even cats and dogs, as well as ducks and geese, are not exempt from this form of diseased liver. Every epicure knows that one of the very daintiest of table delicacies is an artificially produced fattily-degenerated goose-liver, *foie gras de Strasbourg*. Not merely does the liver of the overfed goose, Smithfield prize ox, sheep, and pig, but even of every portly gourmand who eats them, become so loaded with fatty matter that it is sometimes able to burn with a flame, and, as shown at page 40, float like a piece of cork on water of a specific gravity of not more than 1003, and when its tissues are examined under the microscope, its hepatic cells are all seen to be crammed full of oil-globules. In order to show that this is no exaggeration, I give, in figs. 30 and

31, representations of the hepatic cells of patients, showing two different stages of the disease.

Fig. 30.



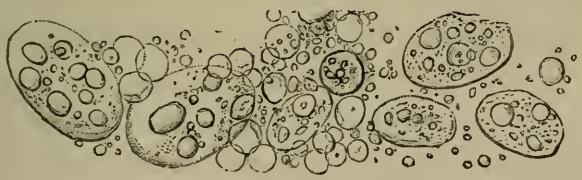
Incipient Fatty Degeneration.

Fig. 31.

Well-marked Fatty Degeneration.

By way of contrast to these, as well as to show how fatty liver occurs in our domestic animals, I further subjoin a woodcut (fig. 32), from p. 195 of my 'Histological Demonstrations,' taken from a drawing made for me by Professor George Brown, of

Fig. 32.



Fatty Degeneration in an advanced stage in the Liver of a Cat, showing the entire loss of the normal granular appearance of the hepatic cells.

the Royal Veterinary College, of the liver of a cat, which he described as having lived luxuriously, spending its time chiefly between eating and sleeping. Although, as a rule, the transformation from normal into diseased liver tissue is slow, this certainly can-

not be said to be the case in fatty degeneration ; for it is well known that a few weeks, five or six at most, suffice to produce the artificially diseased liver of the goose. All that is required to do so being to keep the animal in a warm and dark room, and cram it almost to bursting, several times a day, with fat-making farinaceous food. And what is still more surprising to the pathologist is the fact that the hepatic cells may actually become in this short space of time so full of fat as to cause the surface of the freshly cut section of the animal's liver to assume a distinctly glistening yellow oily tinge, even when viewed in an ordinary light by the unaided eye.

Fattily-degenerated human livers sometimes attain a huge size, twelve or even fifteen pounds being no uncommon weight. Yet notwithstanding this great alteration in their dimensions, from their retaining their natural form and bile-secreting power, they give rise to neither dropsy nor jaundice.

Paradoxical though it may appear to be after what has just been said regarding the association of obesity with fatty degeneration of the liver, I have to call attention to the curiously anomalous fact that fatty enlargement of the liver is an exceedingly common pathological condition in phthisical patients, even when they are emaciated almost to skeletons. This is, however, in my opinion, readily accounted for, when it is known that almost all the cases of fatty

liver that have been met with in persons who have died of phthisis have been found in individuals who have been in the habit of taking cod-liver oil as a remedy for their tuberculous affections. Therefore the rationale of the etiology of the fatty liver in the phthisical appears to me to be easy of comprehension, when it is recollected that, as explained in the physiological part of this work, it is one of the chief offices of the healthy lungs, during the act of respiration, to enable the fats in the blood to become oxidised, and thereby fitted for the purposes of liver transformation and bodily assimilation. Whereas, in the phthisical, the diseased lungs are incapable of performing this part of their functions properly, and the fats of the blood, being only partially oxidised in consequence of the imperfect action of the tuberculous lungs, are not sufficiently prepared for the hepatic cells to be able to transform them into normal assimilable products. Hence, as a simple physiological result, the excess of fatty matter which the hepatic cells are unable to transform and excrete as assimilable material remains behind in them, and encumbers them in the form of oil-globules. Moreover, as a fresh supply of imperfectly prepared fats is brought to the cells with each fresh dose of oil taken, their store of oleaginous material goes on augmenting until there is at length brought about the exaggerated morbid histological condition of hepatic

tissue to which is applied the name of fatty degeneration. In this case (that of the phthisical) the degeneration of the liver cells may, it is seen, be regarded as a simple mechanical process of accumulation, brought about by a defect in the normal pulmonary fat-oxidation process. For a precisely similar reason (the defective preparation of the fatty foods), though with a diametrically opposite result, does the gradual disappearance of fat from the other tissues of the phthisical patient take place, and, as a consequence thereof, a general emaciation of the whole body. My explanation of the phenomenon is this. The fatty matters of the food failing to be properly oxidised by the diseased lungs, the hepatic cells are unable to further prepare them for the purposes of general assimilation. Consequently the excess of fat over and above what remains behind in the liver cells is transported by the circulation to the various organs and tissues of the body, in a condition quite unsuited to their requirements. The tissues, from being consequently unable either to use or store them up, allow them to pass away as so much foreign and useless material, and a general emaciation is the result. These views I of course present to the reader as a mere hypothesis, which he can accept or reject at his own discretion ; for they are only tenable so long as a better hypothesis cannot be substituted for them. (See remarks at page 63.)

From fattily-degenerated livers being occasionally met with in cases of women dying after prolonged suckling, hyperlactation has been thought to be an exciting cause. Dr. Sinéty says (Comptes Rendus, 1873), that in the livers of suckling women an excess of fat-globules is deposited in the cells immediately surrounding the central veins. This, he believes, is a condition contrary to what occurs in ordinary cases of fatty degeneration, where (he affirms) the cells at the periphery of the lobules are those most prone to fatty degeneration.

I have been more than once asked whether or no the period of life has anything to do with fatty degeneration of the human liver, and my invariable reply has been Yes—from the fact that all men and women approaching the period of middle age, as it is called, show a greater or less tendency to obesity. Women are said to lose their 'waists;' men to get 'corporations.' But I ought at the same time to add that fatty livers are frequently met with in children, sometimes only one or two months old. This diseased state must not be confounded with the so-called 'fatty liver of infancy,' which is a normal physiological condition. The proverbial big bellies of children are well depicted in the paintings of Rubens. They are caused by the liver being in infancy equal to one-eighteenth of the weight of the whole body. (See page 41.)

Having said that jaundice is generally absent in fatty liver, I have to call attention to the yet stranger fact that sometimes it appears as if no bile, or at least no green bile, were secreted. Frerichs has recorded a case of the kind, where, at the *post-mortem* examination, the gall-bladder was found empty, and the bile-ducts lined with pale-grey mucus. Similar appearances have been met with in cases of amyloid degenerations of the liver. And although these cases must be regarded as due to a suppression of the biliary functions, yet, strange to say, in some of them no jaundice has occurred. For neither has the skin been more than sallow, the urine high-coloured, nor the stools of a pipeclay hue. Indeed it seems as if a total arrest had taken place, not only of the secretion of bile by the liver, but likewise of the normal transformation of red blood pigment into green biliverdin in the general circulation. A point to be further alluded to when on the subject of white bile in the chapter on diseases of the gall-bladder.

Symptoms of Fatty Liver.

After what has just been said regarding the etiology and pathology of fatty livers, the reader will have learned sufficient facts to enable him to suspect, if not even to detect, the existence of a fatty liver in certain aged and constitutioned individuals. He must remember, however, that the existence of a fatty liver is not always easily diagnosed ; although

I think I may safely say it seldom presents any very formidable difficulties, from the fact that a big, painless, normally shaped liver, associated with a sluggish biliary function, is nearly certain to be the possession of the proprietor of a fatty liver. Moreover, as I have already pointed out, there are three distinctly different-constitutioned individuals who are more or less prone to have fattily-degenerated livers. Firstly, the overfed and obese ; secondly, the phthisical treated with cod-liver oil ; and thirdly, women suffering from the effects of hyperlactation.

Treatment of Fatty Liver.

The best treatment for fatty liver, whether it be in the child or the adult, is regulated diet. Animal foods, with little oleaginous matter in them. The lean of *fat* meat is what ought to be given, not the scraggy, flavourless, nutritionless meat of half-starved animals, but the lean of well-fed, plump individuals.

Next, the avoidance of sugar and sugar-forming foods, such as those belonging to the farinaceous group, which all contain a large quantity of starch, which is no sooner taken into the system than it is first transformed into sugar by the salivary and pancreatic secretions, and then by the liver into fat. French gluten bread, bran bread, and toasted bread may be eaten. Potatoes are to be avoided. For similar reasons malt and spirituous liquors are to be eschewed.

Regular daily exercise in the fresh air is to be enjoined. The hot-air bath and the vapour bath are both useful. The former in the shape of the Turkish, the latter in the form of the Russian bath. Both of which, when judiciously employed, are of immense service in fatty liver.

Lastly, there are certain remedies which are exceedingly useful. The first is the sulphate of soda, either in the ordinary form of Glauber's salts, or in the more pleasant granulated effervescing form, in one or two drachm doses, taken in half a tumbler of hot water, early in the morning, on an empty stomach. The next remedy that I recommend is the carbonate of ammonia, in from five to ten grain doses thrice a day. I was led to use this remedy from noticing the remarkable effects the carbonate of ammonia has upon pigs when given to them in large doses, as I saw done experimentally by M. Kuhlmann in France. Not only did the animals become lean, but their muscles became hard—so hard, indeed, sometimes, that even when well cooked they were quite uneatable.

It is really astonishing to see how rapidly an enlarged fatty liver sometimes decreases under the above system of treatment. On one occasion a married lady, aged 36, whose liver was enormously enlarged, was sent to me by Mr. Fry of Yorktown. I guessed it at nearly twenty pounds ; for though only 5 feet 2 inches in height, she measured 64

inches round what she was pleased to call her waist. I rigidly dieted her—gave her a dose of sulphate of soda every morning, and carbonate of ammonia thrice a day, and to my surprise when I next saw her, which was in exactly a month, she had diminished in girth $11\frac{1}{2}$ inches ! and in four months later $5\frac{1}{2}$ more, making in all 17 inches decrease. Alas! however, for the credit of intellectual humanity, this poor lady relapsed into evil habits, and her liver gradually increased until it attained immense proportions, more than half filling her enormously distended abdominal cavity. The actual girth of which, unfortunately, I do not now know, from my having lost the piece of tape giving it exactly ; but this much I do know, and that is that for some time before her death it was simply prodigious.

According to my experience the majority of cases of fatty liver occur in the persons of patients who have not sufficient moral courage to restrain their vicious dietetic propensities in eating as well as in drinking. For it appears to me that fully one half of the patients I see with fatty livers kill themselves by over-indulgence in food and drink.

Amyloid Liver.

The amyloid form of degeneration of the hepatic tissue has, unfortunately for pathological perspicuity, many aliases—the disease having been by different

writers described under the titles of 'albuminoid,' 'amyloid,' 'cholesterin,' 'lardaceous,' and 'waxy' liver, just as the organ happened in any particular case to present to the eyes of its describer more of the appearance of one than of the other of the substances after which it was named. The above-mentioned different names have, unfortunately, been given to merely different histological stages of the same affection. To prevent ambiguity, I shall therefore only employ the title of amyloid, which I prefer to any of the others solely on account of the morbid deposit presenting in by far the majority of cases a closer histological and chemical resemblance to amylaceous matter than to anything else.

Pathology and Etiology of Amyloid Liver.

A freshly-cut section of an amyloid liver may be described, in so far as its naked-eye appearance goes, as a clear, glistening, pellucid, homogeneous substance, which, although possessing a distinctly greasy-like shiny lustre, is quite distinct from the shiny, oily look of a fatty liver.

In chemical composition it is, though a true animal albuminoid, closely allied, in many of its characters, to vegetable starch. For example, it is stained bluish by iodine. Differing from starch, however, in so far as the blue tint is only developed after the addition of sulphuric acid. While

a mere aqueous solution of iodine in iodide of potassium produces the blue colour with all true vegetable amyloids. An aqueous solution of iodine gives an evanescent reddish-brown tint to amyloid liver substance, instead of the ordinary yellow developed when applied to normal hepatic tissues.

The subjoined comparative table of some of the reactions of chemical substances upon animal amyloid, cholesterin, and vegetable starch, shows their distinguishing characteristics very plainly, and will probably be found of use to beginners in the study of amyloid disease.

Comparative Table of Amyloid Tests.

	Starch	Amyloid	Cholesterin
Water .	Dissolves on boiling	Dissolves on boiling	Unchanged
Ether .	Insoluble	Insoluble	Dissolves
Heat .	Dries up	Dries up	Melts
Sulphuric acid	Chars	Swells up, reddish-brown	Passes through a variety of colours, green, blue, &c.
Iodine .	Becomes blue	Turns only blue after the addition of sulphuric acid. Which blue colour an excess of acid again destroys.	Remains unchanged
Sulphate of indigo	—	Amyloid tissue soaked in it becomes of a brilliant blue; while with ordinary liver tissues the blue fades to a pale green	—

Bock and Hoffmann assert (Virchow's Archiv, vol. lvi.) that the brown and other colours produced

by iodine and iodide of potassium, ranging from a yellowish-brown to a dark sooty black, are entirely due to the varying proportions of the glucogen which chances to be stored up at the time in the hepatic cells.

In a report on amyloid disease made by Dr. Marcet, and published in the Pathological Society's 'Transactions' for 1871, it is said that there is a great deficiency of potash and phosphoric acid, and an excess of soda and hydrochloric acid, in amyloid degenerated organs.

Like the fatty, the amyloid degeneration of the liver tissues may take place not only in childhood, but in early infancy; children have died from it even before they were a month old. This has been particularly noticed as occurring among victims of inherited syphilis, even where no distinct gummatous growths have formed. As the subject of syphilitic livers has been already considered (p. 932), I shall merely here speak of cases of pure amyloid degeneration, which are not at all uncommon in scrofulous, tuberculous, and rickety children, who are at the same time badly fed and badly clad. While saying this, however, it is necessary to add that the children of the well-to-do and even wealthy are by no means exempt from the disease; although they are attacked with it less frequently than the children of the poor.

It is natural enough for us to expect that amyloid, like fatty degeneration of the liver, should be frequently met with in early childhood, when it is

remembered that the hepatic functions are much more active in early than in later life, and that, by a great pathological law, the liability of an organ to disease increases in direct proportion to the activity of its normal functions. The amyloid form of degeneration of the liver materially differs, however, in its pathological history from all other hepatic degenerations ; at least in so far as it is a frequent sequel to long-standing and exhausting suppurating and cachectic affections, such as necrosis of the bones, hip-joint disease, pyelitis, &c. More especially when they occur in an hereditary tuberculous, scrofulous, or syphilitic-tainted constitution.

Signs and Symptoms of Amyloid Liver.

Children affected with amyloid disease of the liver, like those affected with fatty livers, have very protuberant bellies, and in addition are usually sallow in complexion and puny in frame, have very irregular appetites, and usually constipated bowels. Although amyloid disease is more difficult to diagnose than fatty liver, yet when it is far advanced there is in general no great difficulty about it ; for in addition to the predisposing history—already alluded to—accompanied by a painless solid uniform [1] enlargement

[1] There are occasional exceptions to the rule of uniform enlargement, for cases of undoubted amyloid degeneration have been met with where, at the *post-mortem*, the liver has presented a nodulated appearance, somewhat like what is seen in cases of cancer and cirrhosis. (See p. 480).

of the liver, and sallow bilious look, there is in most instances a concomitant amyloid enlargement of the spleen, and also disorder of the urinary secretion from the kidneys themselves partaking of the disease. For be it remembered, amyloid is a constitutional much more than a local affection.

The urine is in general loaded with urate deposits, is usually very scanty, dark-coloured, and of high specific gravity. Yet it may contain albumen, and that too in a quantity which to the uninitiated might appear as being quite out of proportion to its specific gravity. (See chapter on Hepatic Albuminuria, p. 793.) And what may appear to him still more surprising is the occasional absence of renal tube casts from this form of albuminous urine.

In addition to these physical signs of disease, there are in general symptoms of impairment of the mental powers, disordered digestion, lassitude, and general malaise. In fact, the patient often describes his symptoms correctly, as well as graphically, in feeling himself, as he says, 'good-for-nothing.'

Treatment of Amyloid Liver.

Amyloid disease of the liver ought to be treated on purely scientific principles, for, as far as I have been able to ascertain, no form of empirical treatment whatever, except it be that of the administration of large doses of chloride of ammonium, has ever been

found to be of the slightest service. And assuredly the *rationale* of the action of the chloride of ammonium in these cases I have never as yet been able to understand, although so many persons have said that they have found it useful that I suppose its action is undeniable. In fact, I have myself found it act well when given in from 60 to 100 grains a day for several days running. However, I have never trusted to its action alone, but invariably treated the patient at the same time upon what I regarded as strictly scientific principles by doing my best to combat the supposed cause of the disease, be it scrofula, tuberculosis, syphilis, or what it might. Added to which, as amyloid disease has the same *rationale* as fatty degeneration of the liver, the amyloid substance being nothing more or less than the transition stage in the physiological transformation of starch into sugar, and sugar into fat, the same dietetic rules are to be enforced on patients suffering from amyloid disease as I said were necessary for patients labouring under fatty degeneration. So, in order to avoid unnecessary repetition, I refer the reader to them. Moreover, as the victims of amyloid degeneration are, in the majority of instances, the scrofulous and tuberculous waifs of poor ill-fed and ill-clad humanity, it is evident that good clothing and warm dwellings are quite as essential to their cure as suitable food and medicine.

Fibrous Growths, Embolisms, and Blood Extravasations.

This is a part of hepatic pathology requiring, I think, complete revision. I do not believe in the occurrence of a true fibrous hepatic tumour, for the simple reason that fibrous growths never develop in cellular structures, and the liver is essentially a cellular organ. The so-called hepatic fibrous growths are, I believe, and I shall now endeavour to show, nothing more or less than the remnants of old blood coagula.

The nearest approach to a true fibrous growth which I can find among the many recorded cases is one described by Dr. Wilks. It was found in the liver of a sailor aged 34, who died apparently from an attack of peritonitis following upon a second tapping for ascites; and Dr. Wilks found the hepatic veins and the vena cava obstructed by a mass of tough yellow dry fibrous tissue, occupying a large portion of the posterior of the right lobe of the liver, surrounding and compressing the hepatic veins at their junction with the cava, and diminishing their calibre. The liver itself was extremely congested from the interruption of its outward circulation. In some parts actual extravasations of blood had occurred, which I regard as a point of great pathological importance when considered in connection with what I have to say on hepatic fibrous tumours. The cause of the fibrous deposit was not ascertained. There

was no history of syphilis, but there were scars in the groins as from old buboes, and also an excavation in the penis, near the frænum, which might well be regarded as proof positive that he had at some time or other suffered from the disease. But whether the fibrous mass was the result of an old effusion of blood or of true inflammatory lymph, or a spontaneous growth, could not be decided. (Pathological Society's 'Transactions,' vol. xiii. p. 122.)

In order to show that many, if not even all the hepatic fibrous tumours met with at autopsies of patients, are the result of blood extravasations either from undoubted embolisms or other forms of directly effused blood, I cite the following cases observed and reported by different gentlemen.

In 1869 Dr. John Murray published the case of a woman, aged 24, whose liver, though pale in colour, had numerous diffused patches of extravasated blood scattered throughout its whole parenchymatous structure, as well as here and there under 'its peritoneal covering.' Nevertheless the tissues of the organ are said to have been found, when examined microscopically, perfectly normal; and consequently had the fibrous masses not been recognised as blood extravasations, they might readily enough have been put down as fibrous growths surrounded by normal tissue.

Dr. Murray further remarks that the only other morbid condition met with besides the extravasation

was the existence of several small biliary calculi in the gall-bladder ; but these of course could not possibly have had anything to do with the blood extravasations in the liver, which indeed were proved to be the direct result of a general constitutional hæmorrhagic diathesis. For there were precisely similar kinds of blood extravasations in the ovaries, in the uterus, and in the left ventricle of the heart itself.

In 1869 Dr. Payne reported the case of a cab-driver, aged 39, in whom the left-lobe branch of the portal vein was filled by a firm adherent thrombus. The trunk of the vein at its point of entrance into the liver was completely occupied by a dark red, apparently recent centrally soft coagulum, with a discoloured outer older surrounding part, adherent to the walls of the vessel. Both the splenic and mesenteric veins contained coagula, and as there was also hæmorrhagic infarctus of the lungs, the case was looked upon as one of general thrombosis, though the cause of this condition was not ascertained The man had had endocarditis, but it had completely passed away before the time of his death, and the only cardiac sign that remained was an extremely weak and rapid pulse—180 per minute. He had, he said, suffered from palpitation of the heart for twenty years. (Path. Soc. 'Trans.' vol. xxi. p. 228.)

I have not the slightest doubt that this was a case of embolism, from knowing that cases of embolic blood extravasations are occasionally, indeed not so

very unfrequently, met with throughout the liver substance. I recollect well seeing the liver of a woman in the *post-mortem* theatre at Vienna, when I was studying there, in which were a number of dark red cherry and chestnut sized masses scattered throughout both lobes of the liver, the nature of which was at first thought to be peculiar, until on closer inspection they turned out to be merely solid blood coagula, which had apparently escaped from minute lesions in the twigs of the portal veins.

In 1863 Dr. Murchison exhibited to the Pathological Society a liver taken from the body of a woman aged 29, who died at the end of the fifth week of enteric fever. The greater part of the organ appeared perfectly healthy, but on the upper surface of the right lobe was a white circular patch about the size of a florin, beneath which was an opaque yellowish mass bigger than a pigeon's egg, with well-defined margins, enabling the mass to be enucleated by the fingers with little difficulty. The mass when examined microscopically, though slightly softer and more friable than normal liver tissue, was found to consist of the same kind of hepatic secreting cells, loaded with oil-globules and fine granular matter. Dr. Murchison thought it a case of embolism, from the mass closely resembling, he thought, similar masses met with in the spleen and kidneys in cases ascribed to embolism. (Vol. xv. p. 132.)

I may now sum up by saying that the more I

look at the subject of liver fibrous growths, the more convinced I become that the vast majority, if not even all of them, are not, properly speaking, fibrous degenerations at all, but merely the post-products of old blood coagula. There is indeed good reason for my taking this unusual view of the pathological and histological nature of the majority of the hitherto described hepatic fibrous growths. Which is that abnormal fibrous tumours only grow in fibro-muscular organs—such, for example, as the unimpregnated uterus—and, the liver being, with the single exception of the brain, the least fibrous organ in the whole body, it in like manner ought to be, with the single exception of the brain, the last organ of the body to have fibrous tumours developed within its substance.

To the above-described forms of hepatic tissue degeneration I might add a number of others: to wit, the so-called adenoid, tuberculous, lymphadenomatous, &c. &c. But as not a single one of them possesses either characteristic signs or symptoms by which their presence might even be so much as suspected during the patient's lifetime, it would be a mere waste of space to say anything whatever regarding them in a clinical treatise like this. And as there are no known symptoms or signs by which either these or hepatic blood extravasations can be diagnosed, I naturally enough refrain from offering any suggestions regarding their treatment.

CHAPTER XXIII.

TRAUMATIC AFFECTIONS OF THE LIVER.

As general practitioners are frequently, and physicians occasionally, called to the bedsides of patients suffering from the effects of mechanical injuries to the hepatic organ, though this is a medical and not a surgical treatise, I must call attention to one or two facts connected with the prognosis of such cases, which cannot fail to interest them; and to begin with I shall offer the following piece of politic advice to the young practitioner, who perhaps at the very outset of his career may be called to the bedside of a patient the victim of severe mechanical hepatic injury—which is: Never give a hasty opinion of the probable result of the accident; because, while severe and fatal mischief may have happened to the liver, and yet the integuments be scarcely so much as bruised, the organ may be severely bruised, lacerated, or punctured, and yet the patient make a good and quick recovery. I shall give examples to prove the truth of this assertion. In the first place

I may mention that it has been recorded by military and naval surgeons, not once or twice, but a dozen of times, that a rapidly fatal injury has been caused to the liver by a solid cannon-ball, or a mass of shell, without the abdominal parietes being so much as penetrated. The only visible external evidence of injury having been ecchymosis of the integument, and yet the liver was ruptured. But what is more extraordinary still is that there may be actually fatal laceration of the liver, and yet no outward sign of injury whatever visible, as occurred in the case recorded by Mr. Partridge in the Pathological Society's 'Transactions,' vol. xi. p. 127, where a cart-wheel produced this kind of lesion in a lad aged 13.

On the other hand, again, it has happened that the whole of the hepatic abdominal parietes have been completely shorn away by direct violence, and the anterior surface of the liver exposed, without a trace of injury on the organ being visible. As these cases are, comparatively speaking, exceedingly rare, I shall cite one which I saw several years ago, at Shaldon, along with Mr. Thomas Brookes.

The patient, a man of 34 years of age, strongly built and perfectly healthy, had the anterior hepatic portion of his abdominal parietes completely shot away by a blank cartridge from a cannon fired off within a yard or two of him. The cartridge sliced away, as it were, the whole thickness of the abdomi-

nal parietes, and left not only the liver completely exposed, but at the same time perfectly intact. I saw the patient within six hours after the receipt of the injury, and he was then in a state of collapse, and consequently could feel nothing. So I took the opportunity of examining the exposed surface of the liver very carefully. It had the normal bluish-purple tint of a newly killed animal's liver. Its serous covering was moist, glistening, and felt warm to the touch ; and from its having been affirmed that the human liver has been known to manifest pulsation, as spoken of at page 54, I did my best to discern any by throwing the light directly upon it, as well as by viewing it from every side and in every direction ; but not a trace of pulsation could I detect. I next pressed my fingers gently but firmly against every part of the liver's exposed surface, but still failed to feel even the faintest evidence of pulsation. This non-success was not due to an absence of cardiac pulsation, for the radial pulse could be distinctly felt. The patient never rallied. His state of stupor became more and more profound, until he expired in about thirty hours after the receipt of the injury.

The human liver has even been forced through the diaphragm, and partially lodged in the right pleural cavity. A case of this kind is reported in the Pathological Society's 'Transactions,' vol. xvii. p. 164. A man, aged 55, was brought to the Royal Free

Hospital so extremely prostrated that he could give no account of himself. His skin was sweating profusely. His pulse 108. His tongue dry and brown. With dulness and friction sounds over the lower two-thirds of the right lung. He died in seven days, and at the *post-mortem* it was found that the whole of the right lobe of the liver along with the gall-bladder protruded into the right pleural cavity through a six-inch-sized opening in the diaphragm. Yet there were neither signs of peritonitis in, nor of ecchymosis of, the abdominal walls. Unfortunately nothing was ascertained regarding the manner in which this extraordinary hernia was produced ; but no doubt it was the result of direct violence, and had probably existed some time, as there was a well-marked constriction of the liver at the point where the edges of the diaphragm were in contact with it.

It is well to know that even very severe injuries of the liver are not necessarily fatal, at least not immediately so. For example, punctured wounds in the liver, unless a blood-vessel has happened to be injured, are even, as a rule, unattended with serious disturbance. Bayonet wounds on the battle-field have frequently not so much as made the wounded soldier, on account of the injury, fall out of the ranks until after the fight was finished. This remark can surprise no one after what I said on the complete immunity from danger following repeated

puncturing of the hepatic tissue by exploring needles and trocars.

Leaden bullets have penetrated into, and been deeply lodged in, the substance of the human liver, and the patient yet lived for months afterwards. In illustration of the truth of this remark I may cite (from the 'British Medical Journal' of January 10, 1880) a case recorded by Mr. Bernays of a pistol bullet having been lodged three-quarters of an inch deep in the substance of the liver of a child for two months before it died. The first notes of the case were published in the 'Journal' of December 6, 1879. After the child had been considered practically well, with the exception of weakness, she died two months after the accident. At the *post-mortem* the bullet was found in the liver, but not at the spot where it pierced the skin. It had glided over the smooth surface of the liver to the right side, where it appeared to have struck a rib, rebounded, and entered the liver to the depth of about three-quarters of an inch. There were about three ounces of old blood-clot. The small cavity where the ball lay was lined with organised lymph. The functions of the liver were well performed to the last. All the other organs were healthy. Mr. Bernays says that the child did not die from the presence of the ball in the liver, but simply from exhaustion through not being able to take sufficient nourishment.

Even an extensive rupture of the liver may unite, and that, too, rapidly. As was shown by the *post-mortem* of a man aged 38, who died in St. George's Hospital, from fracture of the seventh cervical vertebra, three weeks after having fallen from a height upon a log of wood. For a rupture of the convex surface of the right lobe of the liver, five inches in extent, was found 'perfectly united, with the exception of at some few points, where the peritoneal coat still remained broken.'[1] The rupture in this case did not appear to have extended very deeply; but from the fact of its being so completely healed, it was impossible to conjecture what its original depth actually was. This case proves that even very severe lacerations of the liver are not to be looked upon as necessarily hopeless.

It is a noteworthy fact that the posterior part of the right lobe is the most general seat of rupture in cases of laceration from a blow or a violent compression of the hepatic organ.

The Digestion of the Living Liver.

It was at one time, and that too even until within my own medical day, thought that as the stomach during life was never known to be digested by its own peculiar secretion, all living animal tissues possessed a specific immunity from the solvent action of gastric juice. Experimental physiology upset this

[1] Holmes's *System of Surgery*, vol. ii. p. 648.

theory twenty years ago, by showing that the tail of a living rat, introduced into a dog's stomach through a fistula, and kept there for half an hour or more, was digested. Exactly in the same way, too, as if it had been detached from the living animal, and introduced as a piece of mere dead tissue into the stomach.

No sooner was this important discovery made than all sorts of wild theories were promulgated with regard to the possible cause of the non-digestion of the healthy living stomach by its own gastric juice. Having at one time paid a great deal of attention to the physiology of digestion, and not being a man constitutionally prone to be carried away by theory, I resolved to make for myself some crucial experiments on the action, or rather, I should say, non-action, of the gastric juice on its own secreting membrane. The most telling of which experiments was the scraping away of the epithelial covering of a part of the mucous membrane of the living stomach of a dog, through an artificial fistulous opening, and watching the result—which was, that the portion of the stomach denuded of its epithelial lining (from its thereby ceasing to be protected by a continually replaced layer of alkaline mucous secretion) fell a prey to the solvent action of the gastric juice, and was digested, just as a piece of dead stomach would have been under similar circumstances.

The conclusion forced upon me from the result of

this and other somewhat similar experiments—which I need not here waste time by referring to, as they were published in the 'British and Foreign Medico-Chirurgical Quarterly Review' of January 1860—was that the stomach is protected from the solvent action of its own acid gastric juice by a continually replaced layer of mucus, which is perpetually being secreted by its epithelial lining during the digestive process. From this the reader will easily understand how gastric juice can digest a living liver, and not be surprised to learn that Dr. Mackenzie found a hole of about three inches in depth in the liver of a patient from whose stomach a quantity of gastric juice had accidentally escaped and come in contact with the hepatic tissue. As Dr. Mackenzie's case is, I believe, the first of the kind that has ever been recorded, and possesses several interesting pathological bearings, I shall quote it almost *in extenso* from his paper in the 'British Medical Journal,' May 8, 1880, which is entitled 'Case of Ulcer of the Stomach penetrating into the Liver.' The patient was a man 'aged 56, ill-nourished and rather emaciated in appearance. His skin was dry; pulse 60; tongue covered with whitish fur. Appetite small; bowels sluggish; abdomen distended. He complained of pain in the right hypochondriac and epigastric regions, increased by pressure and followed by sickness. Had been subject to "sick attacks" for years. He was treated with nitro-muriatic acid, small doses of ipecacuanha wine, and

opium pill to give sleep. He improved so much that he was able to go out and superintend a small farm. On April 10 an indigestible supper reproduced the old symptoms, but in an aggravated form. The treatment was continued with varying success up to the middle of July, when he died unexpectedly.

'*Post-mortem eight hours after death.*—A distended stomach concealed all the other viscera; the walls were so thin that the scratch of a finger-nail instantly ruptured them, disclosing the contents to be a coffee-ground like fluid, in quantity about two and a half pints. On raising the stomach, it was found to be attached to the left lobe of the liver, and a large ragged ulcer, opening direct into the liver, was found near the pyloric end. The cavity in the liver contained the same grumous fluid as the stomach, the edges of the ulcer being firmly agglutinated round the orifice, forming a continuous structure. The cavity in the liver had no wall, but was a "burrow" caused by the action of the contents of the stomach.'

The ulcer of the stomach had evidently tried to close itself up by attaching itself to the liver. This effort of nature at repair must have occurred some time before the patient's death, as the edges of the ulcer were firmly adherent to the structure of the liver, and the gastric juice on coming in contact with the exposed inner portion of the hepatic tissue had digested it as if it were a dead organ.

CHAPTER XXIV.

HEPATIC ASCITES.

Ascites, like jaundice, though a frequent, is by no means a constant associate of hepatic disease.

Etiology.

The term ascites simply means a collection of fluid in the peritoneal cavity, the mechanism of its pathology being in all cases of hepatic disease invariably the same. No matter whether the liver affection inducing it be a benign growth or a malignant cancer, the fluid is simply blood serum, and the cause of the serum's exuding from the capillaries into, and collecting in, the peritoneal cavity, is a mechanical obstruction to the normal upward flow of the abdominal blood through the inferior vena cava. The blood being pent up in the abdominal veins, its most fluid part, the serum, exudes through the thin coats of their capillaries by a process of osmosis, collects and becomes pent up in the shut serous sac formed by the peritoneum.

Ascitic fluid thus regarded is seen to be nothing more or less than *pure blood-serum* in an abnormal situation. Its specific gravity, like serum withdrawn directly from blood itself, is consequently 1012.

Signs and Symptoms of Hepatic Ascites.

In all cases, no matter however severe may be the form of the hepatic disease, the effused liquid is at first limited to the abdominal cavity, and then presents us with what may be called a case of ascites pure and simple. Should, however, the obstruction to the upward flow of the abdominal blood be prolonged as well as severe, the effused serum goes on increasing (after the peritoneal sac is quite full) and diffuses itself in the subcutaneous cellular tissue of the lower limbs, thereby producing what is technically known as dropsy. When the effusion in the lower extremities is very great, the exudation extends to the external genitals, and produces œdema of the prepuce and scrotum in men, and of the labia majora in women. In the case of men with phimosis, sometimes giving to the penis a most extraordinary contorted sausage-like appearance.

Difficulty of breathing, with palpitation, may occur in a severe case of hepatic ascites, the cause being merely that the pent-up abdominal fluid pushes the diaphragm upwards to such an extent as to interfere with the free action of the lungs and heart.

Whenever the exudation extends to the tissues above the diaphragm—that is to say, whenever there exists œdema of the thorax, arms, or face, or an effusion into the pleuræ or pericardium—the case is not one of uncomplicated hepatic disease, but one in which the kidneys, heart, or lungs are likewise disordered.

In the later stages of ascites from hepatic disease, it almost invariably happens that the kidneys sympathise with the liver affection, and their function also gets out of order; but that solely arises from the circumstance that the kidneys, as previously pointed out, are the organs to which are vicariously assigned some of the functions of the liver. Hence it is that when the liver functions have been long in abeyance, the kidneys gradually get out of gear, and ultimately break down from the prolonged excess of work they have to perform in daily eliminating a heterogeneous mass of abnormal hepatic materials.

This state of things is not to be confounded with a case of concomitant though entirely independent renal complication, or it will give rise to grave errors in the line of treatment, by leading the practitioner to treat the effect instead of the cause—the kidney instead of the liver—and thereby only aggravate instead of, as he intended, ameliorate the patient's sufferings. I put great emphasis on this point, from well knowing that physicians of undoubted and well-merited reputation occasionally fall into this error, to

the great disadvantage of their patients, as I have shown happened in the case referred to at page 794.

As yet I have been speaking merely of cases of hepatic ascites of easy diagnosis; but, alas! all are not equally easy. Ascites may exist from liver disease, and yet its cause be exceedingly obscure. Indeed, it is sometimes almost impossible for even one well versed in liver cases to decide on the spur of the moment (as it occasionally happens a man in consulting practice is expected to do) whether the accumulation of fluid in the abdomen is due to liver, kidney, or heart affection. Consequently I shall here call attention to a few data which have more than once done me good service in doubtful cases; for if they have helped me to arrive at a correct diagnosis, they will, no doubt, under similar circumstances, prove useful to others.

When called in consultation to the bedside of a patient with a defective clinical history, who is too ill to admit of being subjected to the ordeal of a critical physical examination, a careful consideration of the following facts is invariably of service.

Hints to aid in the Differential Diagnosis of Dropsies.

a. Neither in renal nor cardiac, but in hepatic ascites alone, are the superficial abdominal veins dilated.

b. In uncomplicated hepatic cases there is no œdema whatever above the patient's waist.

c. Albumen may be present in the urine in cases of hepatic, as well as of renal and cardiac disease.

d. If an average specimen of the twenty-four hours' urine be albuminous, it is never, in an uncomplicated case of hepatic disease, of a lower specific gravity than 1010, and very rarely even below 1016.

The reason why, although the urine is equally albuminous, it is not of such low specific gravity in hepatic and cardiac as in renal cases, is simply, I believe, from the fact of the kidney structures being sound, they are able to eliminate the urea and other normal urinary solids. The albumen passing through them solely as an excrementitious substance, from its not being properly metamorphosed by the liver (as explained in the physiological chapter) and thereby fitted for tissue assimilation. Consequently it is eliminated as a foreign material, and not on account of the kidneys being diseased.

e. Even the mere colour of the urine is a valuable diagnostic sign, from the fact that in hepatic and cardiac disease it is never of a lighter shade than normal. In general much darker. While it is usually very pale or smoke-coloured in renal cases. So that even its naked-eye appearance may be regarded as a guide of some importance in making a differential diagnosis.

I take no notice here of the differential diagnostic

help afforded by the employment of the microscope, being now speaking merely of a rough and ready way of arriving, at the bedside of a patient, at a correct diagnosis in doubtful cases. If time and opportunity permit, as a matter of course the microscope should be called to aid, as well as chemical reagents, and they will either negative or confirm the idea of the case being one of hepatic origin.

f. When, in addition to the urinary indications above alluded to, there is present the additional fact that no dropsical symptoms exist above the patient's waist, the suspicion of the case having a cardiac origin is at once negatived.

Hepatic Ascites in Children.

Although it is not a common thing to meet with cases of ascites as the result of liver disease in children, such cases are by no means so exceedingly rare as the majority of persons suppose, from the simple fact, I believe, that the ascites of liver disease in infancy is usually mistaken for that of renal disease, in consequence of the urine in the majority of instances being albuminous.

In 1871–2 I was asked by Mr. Wakefield to see a case of kidney disease. The patient, an intelligent boy of about 9 years of age, had been born at a hilly and healthy station in the Madras Presidency, where his father's regiment was stationed, but he had been

in England for three years, and his disease only began about ten months before I saw him. The chief signs were albuminous urine, associated with an *enormously* distended ascitic abdomen. The navel, from having been originally somewhat imperfectly closed, presented a strange appearance, from the fact of its having its loose cutaneous covering distended with fluid, which projected it for over two inches, and made it look exactly like a Cambridge sausage.

The liver itself was greatly enlarged, nearly half filling the abdominal cavity. The mere look of the urine, but particularly its specific gravity (as I related in the case of suspected kidney disease at page 794), at once showed me that we had a case of hepatic and not one of renal ascites and albuminuria to deal with. Knowing from this that the tissues of the kidneys were not disorganised, I recommended the administration of a powerful diuretic mixture, consisting of squills, digitalis, iodide of potassium, nitrate of potash, and infusion of broom-tops. This for a short time had the desired effect, and greatly diminished the ascites ; but in little more than a fortnight it was as bad as ever. In order to relieve the discomfort the child endured from the great accumulation of fluid in the peritoneal cavity, I proposed tapping as a palliative. The mother, however, would not agree to it, so still stronger doses of the diuretic were had recourse

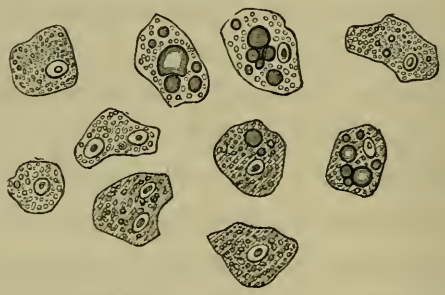
to, coupled with an iodine embrocation over the abdomen. I saw the child once again. He was then greatly improved in every way, and the fond mother thought her boy was in a fair way to recovery; but, alas! I knew too well that it was but a temporary lull in the storm, that the hepatic disease was incurable, and that, ere a few weeks had glided by, her beloved child would inevitably have passed life's Rubicon. As, however, no good could arise from undeceiving her, I left her in the sweet delusion. The child steadily improved for a few weeks more; then suddenly a change for the worse took place; the ascites increased, and the boy died.

As Mr. Wakefield, though he loyally followed my advice, still adhered to his original opinion that the albuminuria and dropsy were due to renal disease, and not, as I had diagnosed, the result of a hardened and congested liver, arising from long-standing chronic subacute inflammation, it was arranged that we should have a *post-mortem* examination, to which the mother readily assented. So I got Dr. Nunneley, my former class assistant, to make it for me along with Mr. Wakefield. The result was exceedingly interesting. For while it confirmed the diagnosis of the case being hepatic to the letter, it revealed the important fact that had the boy been tapped, and tapped frequently, it is not in the least degree improbable that he might have survived for years. For, strange to

say, the liver tissue was but slightly diseased, being merely a little fatty, though greatly congested. So congested, indeed, that when cut into, Dr. Nunneley told me, the blood literally gushed from its section.

The specimen of liver that he brought away and gave me for examination presented a condition of affairs I had never in my life before seen. The whole tissues of the organ—lobular and interlobular—were literally gorged with blood. While the hepatic

FIG. 33.



Human Liver-cells containing oil-globules from a well-nourished child.

cells contained no more oil-globules in them than are often to be met with in well-nourished children.

From these facts I think it is highly probable that had the constant backward pressure exerted by the presence of such a large quantity of ascitic fluid, pent up in the peritoneal cavity, but been by repeated tappings removed from the hepatic blood-vessels, the organ might have regained, perhaps, sufficient vigour to have gradually recovered from its hyper-congested condition. Whereas, left as it was

to its own devices, the congested liver primarily caused the ascites, and the ascites in its turn reacted in keeping up a condition of hepatic hyper-congestion.

The *post-mortem*, therefore, taught me the important lesson, never again to allow myself to be overruled, by either parent or practitioner, until they have had the value of tapping, in such a case as the above, placed before them in the clearest and strongest possible light; for had I known then what I know now, I think I should have been able to win over to my views both Mr. Wakefield and the boy's mother, and thereby perhaps have been able to keep the poor little fellow alive for many months, if not even for several years.

I cannot quit the subject of hepatic ascites without saying a few words in favour of the old-fashioned and nowadays much-neglected drug, elaterium. Although it acts but little on the kidneys, it has a powerful effect in getting rid of ascitic fluid, in consequence of its producing copious watery evacuations. It is best given in the form of pill, and if it be fresh and of good quality, it is seldom or never necessary to give more than one-eighth of a grain in order to produce profuse aqueous stools. Its only disadvantages are its depressing effects; but these are easily combated by a few doses of carbonate of ammonia dissolved in sweet spirits of nitre.

In cases where the œdema of the limbs or exter-

nal genitals is extreme, I generally order small trocars to be introduced into, and kept for fifteen or twenty minutes in, the cellular tissue of the parts. Puncturing with needles is seldom sufficient, and if care be used to employ clean trocars moistened with carbolised oil, there is not the slightest danger of the operation being followed by the disagreeable consequences which one occasionally hears spoken of. At least, all I can say is, that in my experience I have never on one single occasion known any untoward result to accrue from it. Even when I have entrusted the operation to men who have told me of their having previously been unfortunate in such modes of treatment. Of course, when I have been told this, I have been particularly careful to impress upon their minds the necessity of employing perfectly clean instruments, and before employing them to dip their points in carbolised oil.

Dr. Southey has invented what is called a capillary drainage trocar, over the bulbous extremity of which a fine india-rubber tube may be drawn, and the canula retained in the limb for many hours by means of a thread and a piece of adhesive plaster.

Instead of using trocars I often simply employ the points of my hypodermic syringe, and the relief obtained from their employment is sometimes quite surprising. As an illustration of this fact I may mention that on one occasion Dr. Silver took me to

see a member of our own profession, who was in a dying state at the Euston Hotel. The gentleman was not only ascitic, but fearfully œdematous all over the body, from heart disease. I lent Dr. Silver the two nozzles of my syringe, and with these were drawn away in the course of the next eight hours several quarts of fluid, which at once relieved all the most distressing symptoms, and was, I believe, the means of keeping the patient alive for many months.

I have already said so much about ascites in the chapter upon chronic atrophy of the liver, and given so good an example of the value of tapping in the case I saw along with Dr. Bannister (p. 481), when more than twelve gallons of ascitic fluid were withdrawn from the patient in thirteen days, that I need not further dilate upon the subject here ; but simply content myself with asking my readers to reperuse the chapter in question, and in addition cite a case which well illustrates the value of the repeated tappings which I have advocated in distressing cases arising from incurable disease of the liver. The case, which is ably related by Dr. George Johnson in the ' British Medical Journal,' August 7, 1880, is as follows :—

A woman aged 36, on her admission into King's College Hospital, was jaundiced, and her urine deeply bile-tinged. The liver dulness extended from the fifth rib to two inches below the costal margin.

On September 27, 344 ounces of bile-tinged liquid were removed by tapping, with great relief from pain and distension. The fluid re-accumulated, and caused great pain and distress. On October 28 again was removed 284 ounces of fluid. After this, the urine became lighter-coloured, and the skin less jaundiced; but the abdomen again enlarged; on November 26, 271 ounces were removed, and the skin and conjunctivæ nearly regained their normal colour. She recovered appetite and strength, and was discharged January 10, 1880.

Readmitted on June 21. The skin and eyes were deeply jaundiced; the urine contained bile; the abdomen measured forty-four inches and a half at the navel. She had pain and dyspnœa from abdominal distension. On the 24th, 303 ounces of liquid were withdrawn, after which the liver was felt with its thin edge two inches and a half below the ribs. Great relief was afforded by the tapping; but, four days after the operation, she had a rigor. Temperature 102·3°; abdomen tender, and again becoming distended. There was occasional vomiting; the pulse became rapid and feeble, the tongue dry; and she died on July 4, ten days after the last tapping.

The necropsy revealed the case to be one of stricture of the common bile-duct. The liver was olive-green and somewhat enlarged. The gall-bladder distended by dark bile to the size of a turkey's

egg. The cystic and hepatic ducts much dilated. The common duct, just below the junction of the cystic and hepatic ducts, was obstructed by a fibrous thickening of its coats. Very firm pressure on the distended gall-bladder caused only a slight oozing of bile through the common duct into the duodenum.

As Dr. Johnson remarks, the main phenomena were deep jaundice, followed by great ascites; the disappearance of both the jaundice and the ascites after the third tapping; the reappearance of jaundice and ascites after an interval of about five months.

The last tapping, when her strength had been much impaired, was followed by fatal peritonitis. The previous three tappings not only afforded great relief, but unquestionably prolonged her life in comfort for several months.

Dr. Johnson thinks that the pathology of the case was that the dilated ducts compressed the portal veins within the liver, thus obstructing the whole portal circulation, and causing the ascites. The temporary passing away of the jaundice and ascites after the third tapping being explained by supposing that, in addition to a permanent constriction of the duct by inflammatory exudation, there was a congested and swollen condition of the lining membrane, which, after removal of the dropsical pressure by tapping, subsided.

Be that as it may, as he says, the satisfactory

result is an encouragement to repeat the operation of tapping when other means have failed to remove the dropsy.

It may perhaps be just as well for me to call attention to the fact that tapping of the abdominal cavity ought never to be spoken of to the patient or his friends as being a trifling operation; for notwithstanding the facility with which the operation is performed, and the rarity of any disagreeable consequences following upon it, fatal peritonitis is occasionally the unfortunate sequel to the operation. On February 14, 1880, Dr. C. J. Nixon presented to the Dublin Pathological Society specimens from the body of a man aged 45, who died after the operation of simple tapping for an ascites consequent on cirrhosis of the liver. Paracentesis was performed, and several quarts of fluid were withdrawn from the abdominal cavity. In a few days a diffuse erysipelatous blush appeared round the site of puncture, general peritonitis supervened, and the patient speedily sank. At the necropsy an abscess was found lying between the peritoneum and the abdominal wall. Perihepatitis was present. The left lobe of the liver was very large, and the seat of cirrhosis. The spleen was cirrhosed to some extent.

I think I ought here to allude to a very simple and readily prepared form of drainage-tube, the invention of Mr. H. J. Roper. The mode of making

it he describes as follows : 'A piece of ordinary india-rubber drainage-tube is slit up at one end ; then turned up, like a coat-sleeve, for a short distance beyond the slit, and passed into the wound by an oiled probe, placed in the axil of one of the branches thus formed. This, I submit, is more efficient—inasmuch as an open canal is, perforce, maintained for drainage ; and simpler—inasmuch as both drainage-tube and probe are always to hand.'

In no case is to be neglected the judicious administration of tonics, even with or without the additional advantages which are derived from the removal of the pressure of the fluid by tapping. The plan usually adopted of relying on diuretics and purgatives alone is, I believe, to be deprecated ; for as a rule it is not the removal of the already secreted fluid which one has to combat, but its re-secretion, and that lies beyond the scope and influence of diuretics, tappings, or purgatives, and only within the province of tonics.

I cannot refrain from calling my readers' attention to an admirable paper from the pen of Dr. Bristowe in the second volume of the Clinical Society's 'Transactions' on the subject of treating ordinary cases of liver ascites by tonics, for its perusal will amply repay the trouble of referring to it.

Drs. Thompson and R. Liveing relate cases in the third volume of the Clinical Society's 'Transactions,'

which go far to show that copaiba is also a valuable diuretic in cases of ascites. Dr. Thompson successfully combined it with quinine and iron in the case of a man aged 60, suffering from emaciation, anorexia, and thirst, with great abdominal distension from fluid from liver disease. In this case the liver was irregular, knobby, and evidently contracted. A more utterly hopeless case, Dr. Thompson said, could scarcely be, 'and yet the man recovered under the use of the copaiba, quinine, and iron.' A diminution of the abdomen and an augmentation of the urine went on concurrently and commensurately; and this, Dr. Thompson thinks, was mainly due to the copaiba, which acted as a steady diuretic.

Although I have not tried it, I may mention that an infusion of the plant called milkweed (*Asclepias syriaca*) is said by Dr. Spurway to possess specific powers in dispelling dropsical effusions.

CHAPTER XXV.

LIVER SPOTS.

In bygone days, and still among a few practitioners of the old school, one hears a good deal said about the diagnostic value of what are called liver spots—which are brown or yellow cutaneous patches, supposed to be the special result of hepatic disease. As modern pathologists have described two entirely distinct kinds of liver spot, and at the same time attached to them not only a different pathology, but a different clinical significance, I shall consider each separately, and give my views of their intrinsic value and import.

Xanthoma, Xanthelasma, or Vitiligoidea.

While on the subject of the signs and symptoms of jaundice, a passing reference was made to the condition of skin which has received the above titles. Not, however, because it is a specific sign of hepatic disease, but because it is an occasional concomitant of some of the severer and prolonged forms of jaundice.

Before going into the pathology of the condition which has received the above titles, I may remark that when they are translated into plain English they simply mean yellow, or 'yolklike-yellow' patches; which, like many other trivial morbid states, acquire a dignified importance in the eyes of some from the circumstance of their possessing high-sounding names.

Etiology and Pathology of Xanthelasma.

The condition described under this title is a more or less white, creamy, dove-coloured, or dark yellow state of cuticle, about the eyes, nose, hands, feet, and scrotum—wherever, indeed, sebaceous glands are large and abundant. The peculiar condition of skin so named has been supposed to be due to a special and inscrutable manifestation of a peculiar and as yet not understood form of hepatic disease. Instead of which, it is my belief that there is nothing whatever inscrutable about it, it being merely the accidental concomitant and natural sequence of a prolonged attack of jaundice—six months or more—in persons with a constitutional tendency to large sebaceous glands and local subcuticular tissue derangements, originating in the following wise.

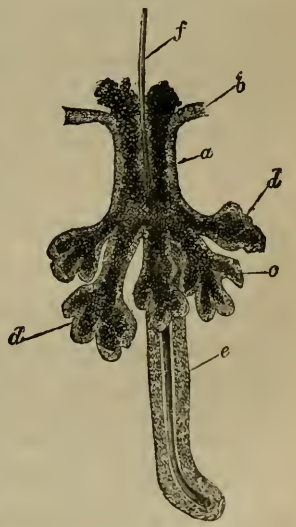
All persons have sebaceous glands about the nose, eyes, hands, feet, and genital organs, which are usually more or less filled with their own normal viscid fatty sebaceous secretion, as shown in the subjoined woodcuts :—

In many persons the glands empty themselves of their secretion with difficulty, in consequence of its being abnormally tenacious; and as a natural consequence the sacs of the glands become distended, and, from bulging beyond the level of the epidermis, give to the skin the appearance of being mammillated or tuberculated—the appearance described as being characteristic of most cases of xanthelasma, barring the yellowness due to the presence of jaundice. Sebaceous glands distended with their own normal secretion may be found on the genital organs of most persons, especially on the penis. Many youths have the sebaceous glands on their faces not only choke-full of secretion, but with an accumulation of dirt in their orifices, causing their faces to look as if they were speckled all over with black points; which black points have received the name of *comedones*. Like all other morbid anatomical formations, enlarged sebaceous glands are often hereditary.

FIG. 34.

A small Sebaceous Gland from the Human Nose filled with its natural secretion, *a*, *b*, *c*.

FIG. 35.



A large racemose Sebaceous Gland filled with secretion (*a*). Mucous lining membrane (*b*). Root branches (*c* and *d*). Hair follicle (*e*). Hair (*f*).

These preliminary remarks, I think, have paved the way for the acceptance of my theory regarding the origin and cause of the cuticular condition learnedly called xanthelasma.

When a so-called xanthomatous portion of the skin is examined under the microscope, nothing whatever peculiar is found in it except an hypertrophy of its connective tissue, enlarged sebaceous glands, and an abnormally yellow-stained sebaceous secretion and cutis vera. In many instances the bile-pigment not only stains the contents of the sebaceous follicles and sweat glands, but actually tints their very walls. Occasionally, too, yellow crystals—erroneously described as tyrosin—are seen in the xanthomatous patches. The crystals are nothing extraordinary, nor can their presence or colour surprise anyone who has had much experience in the examination of healthy sebaceous follicles or even ordinary fat-cells. The fat-cells of mutton, for example—especially during the cold winter months—have often beautiful stellate crystals of margarin in their interiors. And as all crystals take up the colouring matters with which they chance to be surrounded, those in xanthelasma are, like the sebaceous secretion itself, deeply stained yellow by the bile-pigment. Hence there is absolutely nothing at all extraordinary about the so-called xanthomatous portions of the skin, its glands, or their crystals. For just as the urine and the sweat

are stained yellow with bile-pigment in cases of jaundice, so in like manner is the secretion of the sebaceous glands. And the reason why the sebaceous secretion is more deeply stained than the sweat is simply that it does not, like the sweat, flow away as soon as it is formed, but remains pent up in the follicles ; and the longer it remains there the deeper and deeper is the colour it assumes by fresh additions of bile-pigment. Hence the longer the attack of the jaundice is, the darker are the xanthomatous patches.

As tending in the same line of argument, I may mention that Dr. Unna has attempted to prove that the so-called comedones, or plugged-up sebaceous follicles, do not always owe their dark colour to the existence in their mouths of extraneous dirt, but to true pigment. Which he says is not only sometimes brown, as well as black, but even occasionally blue, giving the reaction of ultramarine.[1]

In corroboration of my view of the pathology of xanthelasma, it may be mentioned that those parts of the body most commonly affected with it are those where, under normal circumstances, the sebaceous glands are found both largest and most prominent —to wit, the face, the penis, and the scrotum.

Cases of xanthelasma palpebrarum have been described by Gräfe and Samisch in the 'Handbuch der Augenheilkunde,' vol. iv. part 2 ; while they have

[1] Virch. *Archiv*, 1880–1.

been equally ably treated by Mr. Jonathan Hutchinson in the fifty-fourth volume of the 'Medico-Chirurgical Transactions.' I must mention, however, that my views of the pathology of xanthelasma as above given and the views of these gentlemen are totally different.

Treatment.

The treatment may be summed up in a few words. Daily brisk cutaneous frictions with a hard rough towel, after the parts have been washed with an aqueous alkaline lotion, such as a table-spoonful of carbonate of soda to a pint of water. And if that be not sufficient, a piece of bent iron, such as a watch-spring, may be applied as a scraper to the affected parts, and the sebaceous follicles emptied of their yellow secretion in that way. The biggest and most prominent of them, again, may be emptied by pressing them between the finger-nails, just as young ladies remove black sebaceous spots from their faces.

Chloasma Liver Spots.

This is a rare form of dark discoloration of the skin, only met with in chronic and severe cases of liver disease. So rare, indeed, is the affection, and so few persons have ever seen it, that not only ordinary practitioners, but dermatologists, have confounded it with an entirely different form of cutaneous disease, namely, pityriasis versicolor; the dark colour of the skin in which is not due, as in true chloasma, to the

presence of pigment in the rete mucosum, but to that of a coloured vegetable microsporon parasite attaching itself to the epidermis. From this it is seen that chloasma and pityriasis, though described in manuals as identical diseases, are nevertheless not so, for they possess an entirely different pathology. Indeed, their only point of similarity consists in the fact of their inducing a darkening of the skin.

The colorations of pityriasis may occur on all parts of the body in patients without a trace of liver disturbance, while those of true chloasma generally appear in patches on the anterior part of the trunk, most frequently in the hepatic region. And under no circumstances whatever are they met with unless there exists at the same time a marked derangement of the biliary function. The nearest approach to chloasma in naked-eye appearances, as well as in histological characters, is bronzed skin; and the only mark of distinction, as far as I know, between the true liver spot and bronzed skin, is that bronzed skin presents, as a rule, a much browner colour than chloasma, and is not necessarily associated with hepatic derangement.

As liver spots have neither a diagnostic nor any other form of hepatic clinical importance, I shall dismiss the subject by merely further remarking that so little is known about their true nature that at a meeting of the Pathological Society in 1881, in the dis-

cussion which followed the exhibition, by Dr. Cavafy, of a patient suffering from what he described as extensive chloasma, one of the speakers, who has given considerable attention to skin diseases, took it for a case of leucoderma (vitiligo) modified by jaundice—which is not in the least surprising, seeing that, as before said, few persons have ever seen a case of true chloasma. Though I have seen abundance of spurious chloasma cases, I never to my knowledge came across but one genuine one. The patient was a middle-aged man, who died in University College Hospital with a long-standing jaundice. He had two irregular-shaped almost greenish-black patches on the abdomen. One extended on the right side from near the margin of the xiphoid cartilage to about an inch below the umbilicus ; the other, more to the left of the abdomen, was of about the size of the palm of the hand.

When examined under the microscope (after the patient's death), the cells of the rete mucosum were found to be full of dark pigment, exactly as seen in cases of bronzed skin (see my 'Histological Demonstrations,' page 200, where a woodcut of these appearances is given) and in the skin of negroes. So the conclusion I arrived at was, that in cases of chloasma the cells of the rete mucosum in certain parts of the body have a greater tendency to get filled with bile-pigment than in others, from a deficiency of their vicarious pigmentary eliminating power, which I have spoken of elsewhere (p. 122).

CHAPTER XXVI.

AFFECTIONS OF THE GALL-BLADDER AND COMMON BILE-DUCT.

The human gall-bladder is subject to several important diseases, which, for the sake of brevity, may be summarily divided into three distinct classes ; though every individual member will require to be separately considered. The first class is that where the gall-bladder's function is annihilated, either from the organ itself being altogether absent, atrophied, or shrivelled up, so that its cavity, and its functions as a receptacle of bile, are obliterated. The second class is that in which it is found distended either with green or white bile, mucus, pus, biliary concretions, or calcareous matter. The third is where the gall-bladder is the seat of adventitious growths, either benign or malignant. Each of these causes of gall-bladder distension I shall now speak of separately.

With the appearance and size of a normal human gall-bladder the reader is of course perfectly familiar. Some are not perhaps, however, aware, that while in a normal state its cavity can seldom contain more

than two or three ounces of bile, when in a diseased condition it has been known to contain no less than 260 ounces = a gallon and a half (!) of fluid.

Absence or Atrophy of the Gall-bladder.

This, the first class of gall-bladder diseases, need not detain us for more than a minute. For from this being a purely clinical treatise, and the symptoms of an absent or an atrophied gall-bladder, even with a total annihilation of its functions, being simply NIL, nothing further need be said on the pathology —either of its congenital absence, or of its accidental atrophy and abolition of function—than has already been said in the chapters on physiology, and the effects produced upon it by occlusion of the cystic and hepatic ducts; for what has already been said is amply sufficient for all practical clinical purposes. So I refer the reader back to the chapters on physiology, and gall-stones in the cystic duct. Only here reminding him that there are not only one, but many species of animals, such as those I have already referred to (pp. 81 and 517), which have no gall-bladders, and that even human beings have been born without a trace of a reservoir of any kind whatever for the bile, and notwithstanding the deficiency have developed and lived till adult life. The absence of a gall-bladder has often been noticed in children, who have died within the first six months of birth,

and that oftentimes, too, without their having manifested the slightest biliary derangement of any kind whatever.

Even in adults complete atrophy and obliteration of its cavity have been met with. Mr. Wood, in 1859, showed, at the Pathological Society, a small nodular-looking mass, not bigger than a large pea, which he described as being the only remains of a human gall-bladder. It contained a minute cavity, communicating with the bile-ducts, which were found to be dilated to double their normal size, and filled with viscid bile. The exhibitor thought the atrophy of the gall-bladder was most probably due to a previous attack of inflammation of the organ.

Gall-bladders distended by Liquids.

This, the second class of disease—that in which the viscus becomes distended and enlarged—is one which is marked by varied and very special symptoms, and consequently it is one of so much importance to the practitioner, that I require to particularise the various morbid conditions which give rise to this phenomenon. All forms of accumulation of fluid in the gall-bladder have long been, and still are, erroneously included under the generic term of 'dropsy,' *Hydrops vesicæ felleæ*, and a more inappropriate term could scarcely be given; for none of them has any characters in common with what the

word 'dropsy' really implies, namely a collection of a watery-like secretion. For not a single secretion ever met with in the gall-bladder can properly be said to be 'watery-looking.' Neither bile, mucus, nor pus having any resemblance whatever to water, except that of being in the possession of the property of fluidity. The sooner, therefore, the term 'dropsy of the gall-bladder' is banished from medical books, the better it will be for the credit of the intelligence of the profession. I for one shall certainly put the term 'dropsy of the gall-bladder' on one side, and describe the diseases usually included under that name each as it ought to be in its own true pathological garb.

Signs and Symptoms of Distended Gall-bladder.

The signs and symptoms of a distended gall-bladder are in general plain enough to interpret. In the first place there is more or less fulness, or even a distinct globular tumour, not only palpable to the touch but visible to the eye, in the normal situation of the gall-bladder ;[1] and when the tumour is asso-

[1] As it has happened that when, after having made a careful examination of the abdomen of a patient, I have said the gall-bladder is distended with probably so and so many ounces of bile, my co-consultant has regarded me with an air of significant incredulity, not only from his being unable to guess at the probable quantity of fluid contained in the distended gall-bladder, but from his inability to recognise the presence of any gall-bladder at all, I have given him a hint which may possibly be equally useful to the reader who is desirous of knowing not only how to

ciated with jaundice, which is, however, not always the case, pipeclay-coloured stools and high saffron-coloured urine, together with the presence of bile-acids in the secretion, the case may at once be put down as one of distended gall-bladder by pent-up biliary secretion, in consequence of an obstruction in the course of the common bile-duct. In none of the other cases of gall-bladders distended with liquid is jaundice at all likely to be present, or, I should rather say, necessarily present; for of course it may be an accidental concomitant of any one of them, in the same way as it might be the accidental concomitant of a broken leg or a stone in the urinary bladder.

Gall-bladders distended with Bile.

When a distended gall-bladder has been diagnosed and the patient is jaundiced, it may, in the absence of any reasons to the contrary, be suspected to be filled with ordinary bile, and to be due to the pre-

detect, but how to be able to estimate the size of a patient's gall-bladder through the abdominal parietes. It is, to commence by acquiring the necessary *tactus eruditus* on his own urinary bladder, in the following wise. Before getting out of bed in the morning, and while lying on his back with his bladder full, gently but firmly rub the hand over the supra-pubic region, and a globular tumour will be felt. Then completely empty the bladder, and repeat the rubbing process, noting the difference. Let him do this two or three mornings running until he is quite *au fait* with the manual sensation of a distended urinary bladder. Next let him proceed to educate himself to detect the quantity of its contents by feeling the organ before and after emptying it of measured quantities of urine, and he will soon learn how to be able to tell not only when a gall-bladder is distended, but what is, within certain limits, the probable quantity of its contents.

sence of an obstruction to the flow of bile into the intestines through the ductus communis choledochus. But after making this statement I must call attention to the fact that neither is the existence of jaundice nor of a distended gall-bladder an absolutely necessary consequence of occlusion of the outlet of the common bile-duct, for the very simple reason that the duct may be completely occluded, and yet the bile find its way into the intestinal canal through an ulcerated or other opening at some point above the seat of the obstruction, and thereby nullify the effects of what, under ordinary circumstances, would produce both a distended gall-bladder and jaundice. Such cases are of course exceptional. For in the majority of instances of obstruction to the normal flow of bile through the common bile-duct into the intestines—no matter whether its cause be a cicatrised duodenal ulcer, the pressure of a cancerous or other form of growth, or an impacted gall-stone—the occlusion of the duct is in general followed by distension of the gall-bladder, and its natural concomitant jaundice. Indeed it is only, I believe, in the very exceptional cases in which a gall-stone ulcerates its way into the intestines from a point above the seat of obstruction in the duct that there is associated with it neither a distended gall-bladder nor jaundice.

When the obstructing cause is of a permanent nature—such, for example, as was the case in the

patient with the cicatrised duodenal ulcer, whose biliary organs are delineated in Plate I. p. 113—jaundice is invariably present ; and, as was said, his gall-bladder was at one time (until it suddenly emptied itself) so enormously enlarged as not only to have been felt, but seen, projecting as a globular tumour, through the abdominal walls. As it is a true saying that forewarned is forearmed, I think it well to guard the reader against the possibility of mistaking an enlarged gall-bladder for a kidney; for Dr. Austin Flint, in his 'Practice of Medicine,' relates a curious case in which not only he, but several others of the physicians to the Bellevue Hospital, took a distended gall-bladder for a floating kidney. In order to avoid yet another form of mistake, it must be carefully borne in mind that prolonged occlusion of the common bile-duct may give rise to such enormous distension of the gall-bladder as to cause it to simulate an ovarian tumour. The enlarged organ not only extending to near, but even beyond, the crest of the ilium, and, but for the co-existence of jaundice and other signs of biliary derangement, being almost of necessity liable to be mistaken for an ovarian or other abdominal tumour, entirely unconnected with the liver.

It was formerly thought that when gall-bladders were so distended as to contain a couple of quarts of bile, and reach down into the pelvis, they were very extraordinary ; but since the publication of Mr.

Gibson's case in vol. xi. of 'Edinburgh Medical Essays,' where 160 ounces, that is to say exactly a gallon, of bile was removed from the gall-bladder, and Dr. Barlow's case of a man aged 54, in whose gall-bladder thirteen pints, or 260 ounces, nearly a gallon and three quarters, of bile were found accumulated twelve days after the occlusion of the common duct caused by a gall-stone ('Medico-Chirurgical Transactions,' vol. xxvii.), little is thought of a gall-bladder containing a quart or two of bile. When the gall-bladder has been long distended with pent-up bile, the fluid portions of the secretion are gradually (more rapidly than the solid) re-absorbed into the general circulation, and the remaining bile, as a natural consequence, becomes thicker and thicker, until it yields on analysis quite a different proportionate result from the normal secretion, as is shown by the subjoined analysis of the bile removed from the case of occlusion of the common bile-duct of which the lithograph Plate I. is given at page 113.

The contents of the gall-bladder in this case were found on analysis to consist in one thousand parts of

Water		694·45
Solids		305·55
		1000·00
Pigment . .	Organic matter	288·99
Bile acids . .		
Cholesterin .		
Soda . .	Inorganic salts	16·56
Potash . .		
Iron . . .		

Whereas the specimen of normal bile already spoken of (p. 792), taken from the gall-bladder of the woman of about similar age, and analysed at the same time, was of a specific gravity of 1020, and contained in one thousand parts—

Water		933·27
Solids		66·73
		1000·00
Pigment	Organic matter	56·73
Bile acids		
Cholesterin		
Sugar		
Soda	Inorganic salts	10·00
Potash		
Iron		

The one specimen of bile is thus seen to contain more than four times as much solid matter as the other ; and when the relative amounts of organic and inorganic substances are compared, as was before said, the curious fact is observed, that the difference in the amount of solids in the two cases is almost entirely due to the change in the quantity of organic matter. The inorganic salts having not even so much as doubled themselves. Whence is this ? Soda, the chief inorganic substance found in bile, we have already shown (p. 70), occurs in the forms of glycocholate and taurocholate of soda ; substances which, as before remarked, are re-absorbed from the distended ducts and gall-bladder into the circulation, from whence they are being constantly eliminated with the urine, which is, no doubt, one of the causes

of the inorganic salts being proportionally in such small quantity in the abnormal bile accumulated in the gall-bladder in cases of obstruction to its exit from the viscus.

As I propose going fully into the subject of the treatment of distended gall-bladders, in order to avoid repetition I shall delay my remarks upon those distended with bile until I have completely finished with all the other forms of gall-bladder distension.

Gall-bladders distended with Pus.

Gall-bladders distended with purulent matter are not only not uncommon, but at the same time they fortunately present certain symptoms which may, and often do, lead to their correct diagnosis.

A suppuration of the lining membrane of the gall-bladder is sometimes the result of the presence of gall-stones ; but unless there is at the same time an occlusion of the cystic duct, the suppuration produces no recognisable symptoms during the lifetime of the patient. From the fact of the purulent matter draining away into the intestines by the ordinary biliary channels, and passing out of the body along with the fæces unnoticed, the existence of gall-bladder suppuration is not even so much as suspected, and remains undiscovered until the *post-mortem* examination reveals it. In the American ' Journal of Medical Science,' of January 1857, Dr. Pepper relates a

case where he found the cystic duct occluded by a false membrane, and in the gall-bladder was no less than half a gallon of purulent fluid tinged with bile.

Mr. George Brown has recorded in the 'British Medical Journal,' 1878, p. 916, the case of a woman who, after suffering from an enlarged gall-bladder for a year with gradually increasing urgent symptoms, had six ounces of pus drawn off from it by an aspirator, without apparently diminishing the size of the tumour. And as the symptoms continued urgent it was twelve days later cut down upon, but unsuccessfully, for the gall-bladder failed to be reached. However, after an attack of violent retching, a pint of yellowish fluid escaped from the wound, which was followed not only by a diminution of the tumour, but by the complete recovery of the patient.

The microscopic appearances of pus are shown in fig. 36 (page 1087).

When on the subject of the treatment of gall-stones, I referred to a case in which an abscess of the gall-bladder was connected with the presence in it of gall-stones. The surgical history of this case, as recorded by Mr. Bryant in the twelfth volume of the Clinical Society's 'Transactions,' is that the patient, a woman aged 53, had enjoyed good health until within five years, when she noticed a small painless swelling on a level with and to the right of her navel. It gradually increased until in two years it

attained the size of a hen's egg, when it was opened by a surgeon and some matter evacuated. The opening never closed, purulent matter continuing to be discharged during three years. A second swelling formed a little below and to the right of the other, which was also opened, and pus escaped and a sinus formed. It was laid open, but did not heal up, and in a short time bile began to be daily discharged. On the sinus being probed for two inches upwards, a gall-stone was felt ; so the sinus was carefully enlarged and the stone removed by means of forceps and a lithotomy scoop. The stone measured one and a quarter inches in length, and three-quarters of an inch in diameter. No untoward symptoms followed its removal ; although about half an ounce of bile escaped daily from the wound, the patient's health steadily improved. Six weeks after the operation a Sayres plaster of Paris splint was applied to keep at rest the abdominal muscles, and within a couple of months the wound closed.

Krumptmann relates ('Centralblatt,' March 14, 1873) that on making an incision into a tumour of the size of a pigeon's egg, two inches below the margin of the right ribs, and two and a half inches from the median line, in a jaundiced man aged 64, who complained of subacute inflammation of the liver, nearly a gallon of pus poured out from it. A fistula established itself, and from seven to eight ounces of

pure bile were daily discharged from it. This went on for a whole year, when the fistulous opening became blocked up by a gall-stone. It was dilated, and in the course of four days twenty-two calculi came away. The patient lived for eight years, enjoying, comparatively speaking, good health, notwithstanding the daily loss to his system of eight ounces of bile. He ultimately died at the age of 74, of inflammation of the lungs. The stools, during the eight years the fistula lasted, were of a greyish-white colour, thereby proving the absence of bile from the intestinal canal, and forcing upon us the belief that the process of chemical intestinal chylification must have been carried on solely by the pancreatic and intestinal juices themselves.

The treatment for all cases in which a gall-bladder is distended with pent-up pus is simply evacuation by means of a trocar, and subsequent washing out of the viscus by a stream of carbolised or aceticised tepid water passed into it through the canula, and a general attention to the patient's health. The carbolised water should be of the strength of 1 of carbolic acid to 100 of water, and of the aceticised of a table-spoonful of vinegar to the half-pint.

Suppurating Hydatids of the Gall-bladder.

Hydatids have not only grown, but suppurated and died, in the gall-bladder. Indeed gall-bladder

hydatids may frequently end in so complete a suppuration, that even at the autopsy the débris of the cyst escapes notice, and the case is, in error, put down as being one of abscess of the gall-bladder. Hydatids sometimes distend the gall-bladder to a very great size. An example of a large suppurating gall-bladder hydatid is reported by Dr. Coley in vol. i. of the Pathological Society's 'Transactions,' p. 272. The case was that of a man aged 40, in whom a tumour, extending below the umbilicus, existed for ten or twelve years. It was hard, tender, and extended downwards to the pubes. It fluctuated. On the seventh day after being first seen it burst and discharged itself at the navel, the discharge consisting of pus, bile, and flakes of lymph. Hectic supervened, the patient died, and as at the *post-mortem* the liver was found perfectly healthy, Dr. Coley put the case down as one of suppurating hydatid of the gall-bladder. The spleen also contained hydatids.

Gall-bladders distended with White Liquids.

That a gall-bladder should be found containing a white liquid instead of a dark green one, has been looked upon and spoken of as a marvellous phenomenon. But, like several of the other generally considered inscrutable hepatic anomalies, I think I shall be able to show that there is in reality nothing ex-

traordinary in the fact of white liquids being found, under certain pathological conditions, in the human gall-bladder. For when the light of modern science is focussed upon their pathology they assume a readily comprehensible and simple form. To begin with, I may mention that I believe that there are two entirely distinct forms of white liquids to be met with. One being a secretion from the gall-bladder itself, the other a secretion furnished to it by the liver. The former, which is by far the most common kind met with, I believe, is simply a true mucous secretion, the latter and rarer form an abnormal species of white bile.

White Mucus distending the Gall-bladder.

It has frequently been observed at necropsies, both of jaundiced and non-jaundiced patients, that the gall-bladder has been found full of, and sometimes even distended with, a glairy dirty-white liquid in cases where there has existed an occlusion in the course of the cystic duct, so complete as equally to prevent the admission of bile into, and the exit of any other liquid whatever from, the gall-bladder. The second class of cases again, though due to a much rarer form of disease (one in which there is no occlusion of the cystic or any of the other ducts), has not been considered to be in the least degree more remarkable than the former. From both having

been erroneously thought to be the direct result of one and the same incomprehensible change having taken place in the function (!) of the gall-bladder. While in reality, as I shall now show, there is nothing whatever amiss with the functions of the gall-bladder in either one or the other case, and the presence of the white liquid in the first set of cases is due to nothing whatever beyond a purely normal physiological process (in so far as the gall-bladder itself is concerned) occurring under abnormal anatomical conditions existing in its cystic duct. While in the second class of cases, I believe I shall be equally able satisfactorily to prove that the presence of the white liquid in the gall-bladder is not due to any abnormality in the anatomical conditions either of the gall-bladder itself or the cystic duct, but to a natural physiological, in the general circulation, having been transformed into an unnatural pathological process. According to these views, I think that the true *rationale* of the presence of the white fluid in the gall-bladder in the first set of cases—that is to say, in those where there is present an occlusion of the cystic duct—is readily explained on the following physiological principles.

Firstly, be it remembered, the gall-bladder does not form, but only stores up, bile.

Secondly, it possesses a mucous lining membrane whose duty it is to keep its interior continually lubri-

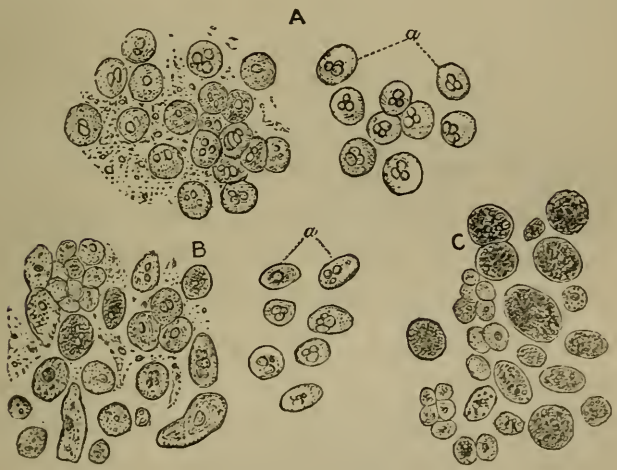
cated with mucous secretion, in order to prevent the stored-up bile acting upon its lining membrane. Such being the normal state of matters, what happens when, from some abnormal cause or another, no bile whatever reaches the gall-bladder? Simply this :—

Should the gall-bladder chance to be full of dark bile at the moment when the occlusion of the cystic duct takes place, it is obvious that this highly-coloured biliary secretion must have been got rid of ere the viscus became filled with a white liquid; and as the occlusion of the cystic duct effectually prevented the gall-bladder emptying itself of the dark green bile by the usual channel into the intestines, it got rid of it in some other manner—namely, by osmosis, through the instrumentality of the capillaries ramifying in its coats. The bile having been simply osmosed into the general circulation, and thence eliminated by the kidneys and skin. Should it, however, chance that the patient died at the precise moment the gall-bladder had got rid of all its biliary contents, then the peculiar phenomenon of an occluded cystic duct and an empty gall-bladder would be found at the *post-mortem*. Supposing, however, that the patient lived somewhat longer, a still further change would then be brought about in the contents of the gall-bladder. From being empty, it would gradually again become filled, but this time not by bile—for

none could possibly find its way into it through the occluded cystic duct—but filled by its own normally secreted mucus. For, from its mucous membrane being in a healthy state, the mucus would be daily and hourly secreted, just as if there was no obstruction in the cystic duct preventing the entrance of bile into the gall-bladder; and, as this secreted mucus could not get out of the gall-bladder, any more than bile could get in, the secretion would go on gradually accumulating within the viscus, until at length it distended it. And if the patient happened to die at this stage, the still more unusual phenomenon of a gall-bladder full of white mucus, instead of dark bile, would, as a natural result, be met with at the autopsy. In proof of the correctness of this explanation of the pathology of at least one of the kinds of white liquids occasionally met with in human gall-bladders, I may briefly quote the report of the *post-mortem* of a woman aged 43, published by Dr. Duckworth in vol. xvii. of the Pathological Society's 'Transactions.' It is there stated that the gall-bladder was found enlarged and full of a transparent thick yellowish mucus, with abundant flaky masses of columnar epithelium and plates of cholesterin suspended in it. The commencement of the cystic duct was firmly plugged up by a gall-stone the size of a musket-ball, the common bile-duct being quite free. There was a distinct history of

the occasional passage of gall-stones accompanied with jaundice. This case so clearly and so completely confirms the above views regarding the way in which gall-bladders get filled with white mucus, that it requires no comments to be made upon it by me. Thus far, then, we have a truly scientific explanation of the strange phenomenon of a human gall-bladder being filled with at least one of the forms of white liquid instead of dark green bile.

FIG. 36.



Pus and Mucus Cells.—A. Pus cells before and after treatment with acetic acid. B. Ordinary mucus cells: *a*. After treatment with acetic acid. C. Mucus cells loaded with pigmentary matter.

The scientific explanation here given, it will be observed, is exactly the same in its major details as that already offered respecting the *rationale* of

cases in which the hepatic bile-ducts are found distended with white liquid in cases of jaundice from suppression, as detailed at page 107. In the cases of the gall-bladders containing white mucus, however, there is, as a general rule, no jaundice whatever, in consequence of there being usually no impediment to the secreted bile flowing directly along the hepatic and common bile ducts into the intestines. As has already been explained in the chapter on occlusion of the cystic duct by biliary concretions at page 604.

White Bile.

It was mentioned in the chapter on fatty degeneration of the liver, that there are good grounds for believing that under certain pathological conditions the formation of green bile-pigment does not take place; for it has been asserted that at autopsies gall-bladders have been met with containing a white fluid which resembled bile in all its chemical properties, except one. Namely, the total absence of biliverdin. A specimen of this kind of colourless biliary fluid, which was taken from the human gall-bladder and examined by Ritter (Comptes Rendus, vol. lxxiv.), contained cholesterin, bile acids, mineral salts, fats, and other biliary organic matters, with the single exception that it contained no biliverdin. To all intents and purposes, therefore, the fluid was true

bile, minus pigment. Had Ritter's remarks stopped here, I should have completely agreed with him; but he has unfortunately supplemented his description of the white biliary liquid with the remark that in such cases there is not only usually present jaundice, but a fatty condition of the liver. This assertion, I think, is not merely a theory, but an erroneous theory; and I believe I can give both a reasonable and a scientific explanation why white bile was found in his non-jaundiced patient's gall-bladder, without regarding the circumstance, as he did, as very extraordinary. Indeed, I shall be able, I think, to show that, contrary to what he imagines, the very fact of the absence of jaundice in such cases is an essential factor to the easy comprehension of the *rationale* of the presence of white bile in a patient's gall-bladder. My theory, indeed, not only logically explains the presence of the white bile, but the absence of the jaundice, in all such cases, and consequently shows that Ritter's own case was no exception to the rule. And, further, it shows that when jaundice is associated with white liquids, either in the gall-bladder or bile-ducts, the fluid is then not 'white bile' at all, but merely the more common form of white liquid, which I have already described as simply gall-bladder and bile-duct normal mucous secretion. White bile, on the contrary, being, as I shall now show, a pathological and not a physio-

logical product, from its being the result of an arrest of the normal oxygenation, and consequent transformation of blood hæmatin into biliverdin in the general circulation. In consequence thereof, from the liver finding no green biliverdin to excrete and pigmentise its secretion with, and the skin none to deposit in its rete mucosum and produce jaundice with, there exists the double phenomenon of white bile and no jaundice. The liver's function itself, in fact, not being disordered at all; but, just as the children of Israel failed to make proper bricks from not being furnished with straw, in like manner the liver fails to secrete properly coloured bile, from not being furnished by the circulation with the necessary pigment for its coloration. As Ritter points out, the WHITE BILE is true bile in every sense of the word, except that it is not pigmented. While the absence of jaundice again, which he considered so peculiar, I equally attribute to the absence of bile-colouring matter from the circulation. There being no biliverdin to stain the skin, as a natural consequence there can be no jaundice and no saffron-coloured urine, though there may be, at the same time, from the same cause, most probably colourless stools. Thus, then, I think, I have now given two logical scientific theories capable of rending asunder the veil of mystery which has hitherto enshrouded the origin of the white liquids met with in the human gall-bladder. Those who

have still doubts of the correctness of the above explanation regarding the absence of colour from the bile, had better carefully peruse what I have said regarding the formation of biliverdin at pages 68 and 119. And as there are as yet no known signs or symptoms by which the existence of white liquids in the gall-bladder can even so much as be suspected during life, I may be excused for declining to offer any hypothetical suggestions regarding their treatment.

Gall-bladders distended with Biliary Concretions.

Gall-bladders may be distended with gall-stones. The stones found in a distended gall-bladder may be not only of all shapes and sizes, but even of different colours and composition. Thereby proving that they have been formed at different times, and under different pathological circumstances.

A gall-bladder may even be completely filled by one large stone. Which may be as big as a goose's egg; and in that case the hard lump can be felt through the abdominal parietes. An excellent specimen, showing how a single gall-stone may completely fill up a gall-bladder (where the stone measures $1\frac{3}{4}$ by $1\frac{1}{2}$ inches in diameter), is among the wet pathological preparations in the museum of the Royal College of Surgeons.

Gall-bladders, though they may be chokefull of calculi, are very seldom, however, found much en-

larged. For the simple reason that as soon as the viscus becomes filled with the calculus or concretions, from there being no more room left for bile to lodge within it, the calculus or concretions, from failing to be supplied with fresh materials for their aggrandisement, cease to grow bigger. This is, however, not always the case, for it sometimes, though but rarely, happens in the case of gall-bladders, as it does with the pelvis of the kidney, that a conical-shaped stone forms at the gall-bladder orifice of the cystic duct, and acts like a valve, more freely admitting of the entrance than of the exit of bile. Just as I showed had occurred in the specimen I exhibited to the Pathological Society ('Transactions,' vol. xv. p. 147), where fifty-nine calculi were found in the pelvis of one kidney; from its having a conical-shaped stone acting as a valve at the mouth of the ureter.

Not only may gall-bladders distended with stones be felt through the abdominal walls, but it has been affirmed by more than one accustomed to manipulate liver cases, that they have detected the actual presence of *numerous* gall-stones in a distended gall-bladder, by their giving origin to a distinct crackling sound, not alone audible by the stethoscope, but recognisable through the sensation of sound given by them to the finger-tips. Be that as it may, there may be said to be but little difficulty in diagnosing the

presence of calculi of any great size or number in a gall-bladder, from the simple fact that firm pressure over the organ, in such cases, is always attended with considerable pain ; and when there are no signs to otherwise account for the pain, and there is reason to suspect the existence of gall-stones, the distension and hardness of the viscus may, with a tolerable chance of exactitude, be put down as a case of enlargement of the gall-bladder caused by the presence in its interior of gall-stones.

Distended Bile-ducts.

Not alone gall-bladders, but even the bile-ducts become enormously distended with pent-up bile.

In the first volume of the 'Dublin Hospital Reports,' Dr. Todd gives the case of a girl, aged 14, who, from an occlusion of the duodenal orifice of the bile-duct, on account of a scirrhous tumour of the pancreas, had the bile-duct so dilated that it reached down into the pelvis.

A distended gall-bladder greatly complicates the diagnosis in cases of suspected distended common bile-ducts ; for they may occur together, or the bile-duct may be even enormously distended and the gall-bladder quite empty. The latter state of things occurs when there exists an obstruction in the cystic duct itself, as well as an occlusion of the common bile-duct, preventing the secreted bile from finding

its way into the gall-bladder. In which case the pent-up bile only accumulates in the common and hepatic ducts, and not in the gall-bladder at all. A case of this kind was reported by Dr. Halliday Douglas in the 'Edinburgh Monthly Medical Journal' of February 1852. The case was that of a maid-servant, aged 17, whose common bile-duct was so distended with bile, that, although she had thirty ounces withdrawn from it by tapping a month before her death, it was found at the *post-mortem* to contain nearly a gallon of bile. Her clinical history was peculiar, for the complaint was thought to have begun three years before her death, that is to say, when she was only fourteen. Her symptoms were pain in the right side, recurring again and again with paroxysmal aggravations and rigors until within three months of her death, when it began to be almost incessant, and the jaundice became much more decided. An acutely tender tumour was felt and seen in the seat of the gall-bladder, and dulness extended five inches in the perpendicular line. Her agony was sometimes intense. The pulse was in general 100. The tongue dry and brown. The bowels constipated, but the fæces dark-coloured. She had profuse perspirations. As the tumour fluctuated, it was tapped with a hydrocele trocar, and thirty ounces of liquid drawn off. This gave immediate relief. She gradually, however, got more

and more emaciated, and died somewhat suddenly about a month after the tapping. At the *post-mortem* examination a large fluctuating sac was found, occupying the whole right side of the abdominal cavity, and closely adherent to the lower surface of the liver. It contained within a few ounces of a gallon of yellow syrupy offensive fluid, in which were crystals of cholesterin in abundance. In the hepatic extremity of the sac were the orifices of the hepatic and cystic ducts, dilated so as to admit the finger. The gall-bladder was undilated. In fact, the whole sac consisted of a dilatation of the common bile-duct, and nothing else.

The non-dilated condition of the gall-bladder, in this case, Dr. Halliday Douglas attributed to the possession by the cystic duct of a valvular orifice.

Treatment of Gall-bladders and Bile-ducts distended by Biliary Concretions, Bile, Pus, and other Liquids.

The treatment of distended gall-bladders and bile-ducts is a point of far greater importance than at first sight appears, from death being the almost inevitable result when they are left unattended to. It is not, however, the mere factor of the distension of the biliary appendages, which, *per se*, causes the death of the patient, but, as will be immediately shown, the combined result of three totally distinct pathological factors, one and all of which

may be said to be a proximate cause of death. Being a zealous advocate of the adoption of the operative plan of procedure in cases of distended gall-bladder and bile-ducts suggested by Petit nearly a hundred and fifty years ago—not alone, however, on new grounds, but with additional facts in its support—I beg leave to remind my reader that in looking upon it with a critical, or, it may even be, with a somewhat cynical eye, it will be well for him to remember that the majority of cases of jaundice from obstruction are much less under the power of remedial agents than those arising from suppression, in consequence of our having three distinct conditions to combat. Firstly,—The derangements originating in the absence of bile from the digestive canal. Secondly,—The morbid effects arising from its accumulation in the ducts, and consequent interruption to the hepatic functions. Thirdly,—The general poisonous action on the system of the reabsorbed bile.

As regards the first of these effects—namely, the derangements arising from an absence of bile from the digestive canal—it may be said that if these were the only difficulties with which we had to contend in cases of jaundice from obstruction, they could easily be overcome. For, in the first place, the mere absence of bile is not attended with any immediate danger. A circumstance which has led to the

common belief that the presence of bile is not absolutely essential to life. Experiments on dogs with biliary fistulæ, like those before referred to, as well as cases of disease in the human subject, have proved that life may be sustained, under certain conditions, for a very long period, without bile reaching the intestines. Indeed, the only immediate bad effects which appear to result from its absence are costive bowels, great flatulence, and extremely offensive stools. While again the indirect bad results—namely, loss of flesh, &c.—as has been proved by experiments on animals, can be counteracted by giving an additional amount of food ; and the direct results of constipation, flatulence and fœtor, may be easily overcome by appropriate remedies.

The secondary morbid effects—namely, those arising from the accumulation of bile in the ducts—are unfortunately not so easily under control. Could we remove the cause of obstruction, these would, of course, immediately cease. This, however, is seldom in our power, except in the case of gall-stones, the expulsion of which, as I have already shown, we can aid in various ways.

When the occlusion of the common bile-duct is caused by an organic tumour, no treatment of ours can be expected to remove the obstacle, and sooner or later the patient is carried to an untimely grave. Our efforts at relief in such a case ought therefore

to be directed into another channel; and here, in order to give the sufferer at least some chance of recovery, even although it be little better than a forlorn hope, I cannot refrain from recommending, in cases of permanent occlusion of the duct in which there is great distension of the gall-bladder, the establishment of an artificial biliary fistula. Dogs, as is well known, live perfectly well for years after the artificial establishment of a biliary fistula in them; and human beings (as I have already shown at page 673, and will yet further show) in whom a fistulous opening into the gall-bladder has spontaneously or accidentally been established, have also been known to live an equal, indeed a much longer, period of time. For example, at page 273 of the first volume of the Pathological Society's 'Transactions,' Mr. Obré relates the case of an exceedingly corpulent lady, aged 75, who about six years before her death observed a globular swelling, of the size of an orange, at the junction of the right hypochondrium and epigastrium, which, after remaining dormant for a time, suddenly became painful and elastic. It was punctured with a knife, and pus and bile flowed from it, and continued to do so, more or less, through a fistulous opening which established itself, until the time of her death five years afterwards. The fluid which flowed away during the five years she lived after the opening was made,

was clear, thin, and of a deep yellow colour. The patient's health remained good, with the exception of being dyspeptic. The bowels were irregular, and the stools pale; though sometimes they were dark in colour and loaded with bile, showing that there was an occasional communication between the bile-ducts and intestines.

At the *post-mortem* examination the gall-bladder was found thickened, and firmly contracted round a gall-stone weighing ninety-four grains. The bile-ducts were all pervious, and the tissues of the liver quite healthy, in spite of the biliary fistula having existed for five years.

A case was reported by Dr. Moxon, in 1866, where a woman, aged 50, after a bilious attack complained of pain and swelling a little below and to the right of the umbilicus, which gradually became as large as a cricket-ball, and ultimately burst, and discharged a sero-purulent fluid through a small opening. After continuing to discharge for a month, pure pus began to come away, and shortly afterwards four gall-stones of about the size of small marbles were found on the poultices. They passed without any pain; were flattened on their sides from apposition, and consisted of cholesterin. The opening did not close up after their discharge, but another stone, and a quantity of friable material, supposed to be dried bile, came away. After which the opening

healed up ; which was not until nine months from the time it formed.

A case of human biliary fistula which discharged eighteen ounces of bile during ten days and then healed up, is reported in the 'Berliner Klinische Wochenschrift' for April 7, 1873, by Dr. J. Hertz. A sempstress, aged 28, enjoyed comparatively good health until she had an attack of enteric fever, a month after which she was seized with severe abdominal pain, soon followed by a swelling the size of a hen's egg, near to and to the left of the navel, which disappeared of its own accord in a few days. Eight months later it reappeared as a sharply defined painless tumour, with a constricted neck. Its lowest portion being about three inches broad. Two months later it fluctuated, and the skin over it was red and tender, like a pointing abscess. An incision was made into it, and about two ounces of a white slimy fluid escaped—not pus. Ten days later a gall-stone was spontaneously discharged ; and during the next eight days thirteen more, varying from the size of a pea to that of a small hazel-nut. Four days later a stream of bile suddenly burst from the wound, and continued to flow from it, at the rate of eighteen ounces a day, for ten consecutive days. From this time all traces of bile disappeared from the stools, and, although she had a good appetite, she complained of weakness ; so an attempt was made to

close the fistula by ligaturing its orifice, treating it like a hare-lip. Six days elapsed after its closure before bile reappeared in the fæces. The patient then regained her strength, and within a month was able to resume work.

It must be remembered, however, that the mere tapping of the gall-bladder would, in a case of gall-stone impacted in the common duct, or indeed in any case whatever of occlusion of the common duct, be of no permanent advantage, whereas the extraction of the gall-stone or the establishment of an artificial biliary fistula would be of the greatest service in prolonging the life of the patient. For what in reality is wanted in a case of enlarged gall-bladder, or ducts distended with pent-up bile on account of a permanent obstruction to its normal mode of exit into the duodenum, is not only to get rid of the accumulated bile, but also of the pernicious results on the hepatic tissue itself of its accumulation. For as soon as these pernicious results are got rid of, the normal biliary secreting function of the liver naturally enough re-establishes itself.

In addition to what I have already said—in the chapter in which I recommended the artificial removal of gall-stones from the hepatic appendages (page 672) and the above facts—as incentives to the production of artificial biliary fistulæ in appropriate cases, as well as the removal of impacted gall-stones by the

surgeon's knife, I may mention that not only one, but several successful cases of emptying the human gall-bladder by operation have been recorded. One was brought before the Royal Medical and Chirurgical Society, in November 1879, by Mr. Lawson Tait, under the title of 'Choleocystotomy performed for Distended Gall-bladder due to the Impaction of a Gall-stone.'

The woman had spasmodic pains in the right side, aggravated by walking and lifting slight weights; presented a cachectic appearance, suffered from headache, sickness, and obstinate constipation. The seat of pain was over the right kidney, where there was a heart-shaped tumour, firm and elastic without fluctuation, tender to the touch, and movable to each side. On August 23, the abdomen was opened in the middle line to the extent of four inches. The tumour was found to be a distended gall-bladder containing a white starchy-looking fluid and two large gall-stones, one lying loose, and the other impacted in the entrance of the duct. The latter was removed. It weighed 6·11 grammes (94·3 grains). The wound in the gall-bladder was stitched to the upper end of the wound in the abdominal walls by continuous sutures; the aperture into the bladder was left open, and the rest of the abdominal opening was closed in the usual way. The operation was performed antiseptically, under ether. The patient rallied completely in a few

hours, and the dressings of the wound were found stained with healthy bile. The flow of bile from the wound continued till September 3. The wound was completely healed on September 9. On the 30th, she went home quite restored to health.

Still further to encourage surgeons to take operative proceedings in cases of dangerously impacted gall-stones, I may mention that Mr. Bryant communicated a case to the Clinical Society in 1879, in which a biliary calculus was successfully removed from the gall-bladder of a woman aged 53, who was a patient in Guy's Hospital under his care in the preceding year with two discharging sinuses of three years' standing, following an abscess, which had been previously forming for two. At first the sinus was laid open, and pus alone escaped; but subsequently, as bile flowed in quantities from the wound, an exploratory operation was performed, and, at a depth of two inches, a biliary calculus, one inch long, taken out of the gall-bladder. Everything went on well after the operation; and although bile continued to escape from the wound for about two weeks, the parts quite healed, and in about four months the patient left the hospital cured.

This case shows that nature might be copied, and gall-stones removed from the gall-bladder through the abdominal walls; and indicates that, under certain circumstances, the operation is justifiable when

the sinuses by their presence are setting up inflammatory and suppurative changes about the gall-bladder, without any obstruction to the bile-ducts, as well as in the more serious class of cases in which the cystic or common bile-duct is obstructed, and dropsy of the gall-bladder, with jaundice, complicates the case.

Dr. J. M. Sims, a still bolder and generally successful operator, cut down upon the enlarged gall-bladder of a lady aged 45, and extracted therefrom no less than sixty small gall-stones, and nearly thirty ounces of fluid which was not bile, but mucus. The operation seems to have been a very tedious one, as it is reported to have lasted one hour and sixteen minutes. Chiefly on account, it is said, of the difficulty experienced in securing the coats of the gall-bladder to the sides of the incision, and closing up the wound. It was performed under the antiseptic method, and although the lady lived only eight days, the immediate benefits of the operation were considerable, as it relieved the pain, nausea, vomiting, as well as the itching, and at the same time produced natural stools. The *post-mortem* showed that there was no peritonitis, and that the gall-bladder, which was already firmly adherent to the abdominal walls, still contained sixteen *sacculated* gall-stones.[1] The cause of death Dr. Sims considered to have been the ab-

[1] *Brit. Med. Jour.* 1878, p. 811.

sorption of poisonous biliary salts. A doubtful theory, for biliary salts are absorbed in every case of biliary obstruction, and do not cause death in eight days or even in eighteen months.

The question of the removal of gall-stones by operative procedure was exhaustively treated in the 'Mémoires de Chirurgie' in the year 1700. In a case there discussed, the stone was withdrawn by the forceps, and the author drew an analogy between it and the operation of lithotomy.

I verily believe that the day is not far distant when the removal of impacted gall-stones from the common bile-duct by the surgeon's knife will not only be a frequent, but as successful an operation as that of the removal of a calculus from the urinary bladder. For just as the presence of urinary calculi can be indubitably ascertained by sounding, so also may the existence of gall-stones be equally indubitably demonstrated. Dr. Whittaker, in a paper in the 'New York Medical Record' (1882), advocates the employment of a long hypodermic needle, as a means of determining the presence of gall-stones and their situation. He relates a case of occlusion of the bile-duct, in which the diagnosis lay between gall-stones and cancer, in which he used the needle repeatedly without any bad effect, and at length, at the depth of 'four and three-fourths inches,' struck a stone. No inconvenience followed. He then operated, and although

the case ended fatally, that is no reason why the attempt should not be made again.

As regards the sounding for gall-stones, I may remark that as I have not only repeatedly inserted darning-needles into the hearts of dogs and rabbits (while studying the rhythm of their pulsations), but kept them there for ten or fifteen minutes at a time without apparently inconveniencing the animal, far less producing disagreeable after-effects, I see no reason whatever to anticipate the slightest danger supervening in probing for gall-stones. And, although I have never as yet done so myself, from my extensive experience in the use of long needles in dogs, &c., I have no doubt the best way to be successful in the search for gall-stones by sounding would be to employ a six inches long fine sharp-pointed steel wire, of not more than the diameter of a darning-needle, fixed in a handle in order to communicate the more readily the sensation of hardness to the hand. Moreover it may be here added that the escape of a small quantity of bile into the peritoneal cavity is attended by merely imaginary dangers. I say this from the fact that bile has again and again been known to escape into the peritoneum after the accidental rupture of the gall-bladder, and not lead to the death of the patient, nor even to so much as peritonitis. In the fourth volume of the 'Medico-Chirurgical Transactions,' a case is actually recorded where thirteen pints (!) of

bile were removed by tapping from the abdomen of a boy whose gall-bladder had been ruptured three weeks previously. He was tapped twice, and after forty-eight pints, in all, of bilious-looking fluid had been withdrawn, he got well.

As, from the histories of the cases previously cited, it is clearly seen that there would be no very great risks run in establishing a biliary fistula in the human subject, I shall now point out what I consider would be its advantages.

By the artificial establishment of a biliary fistula the human being would be placed as nearly as possible in the same condition as the animal in which the operation has been performed for physiological purposes, and, we might reasonably hope, with an equally favourable result, at least in as far as the biliary functions are concerned. In the first place, we should have removed all the derangements resulting from the interruption to the flow of bile, and consequent upon the distension of the ducts. In the second place, we should have obviated the danger arising from the poisonous effects of the re-absorbed bile, which are of no trifling nature ; and, lastly, we should only require to combat the evils arising from the absence of the biliary secretion in the digestive process, which, as was before said, can to a certain extent be overcome by giving an additional quantity of food, and paying attention to the bowels. In these

remarks I have omitted taking into consideration the effects that might arise from a tumour, or other obstructing cause to the biliary secretion, for these would in no way be directly influenced by the mere establishment of the biliary fistula. As might naturally be expected, the patient would, like animals with artificial biliary fistulæ, lose flesh, become emaciated and weak, his hair have a tendency to fall off, his bowels to become irregular, and a great and an almost constant discharge of foul-smelling gases take place from the intestinal canal. At length, after a shorter or longer period, he would sink and die, unless the fatal termination were retarded by giving him pig's bile in capsules, and at the same time allowing him an additional quantity of nourishing food. For death from want of bile is nothing else than death from slow starvation. The fact regarding the beneficial effects of an additional quantity of food in prolonging life should never be lost sight of in the treatment of any case of obstruction of the gall-ducts ; for, by attending to this circumstance alone, it is often in the power of the medical man to keep his patient alive for a considerable length of time. Such then being the case, if a sufficient artificial supply of bile be at the same time given I can see no reason whatever for the patient not surviving for a long period of time.

The artificial establishment of a biliary fistula in

the human subject is therefore not such a Utopian idea as might at first be imagined. Distended gall-bladders having been several times tapped, as we see with success, both in this and other countries, the permanent establishment of a fistula, if done in the manner I shall immediately point out, would even, in my opinion, not be a more hazardous operation than simple tapping. Biliary fistulæ in dogs are generally made in a single operation, by cutting through the abdominal parietes, seizing the gall-bladder, stitching it to the lips of the wound, and inserting a canula. But here there is always some danger of the wound not healing by the first intention, and of the passage of bile into the abdominal cavity. In the case of the human subject I should therefore recommend the inducing of an adhesion of the gall-bladder to the abdominal parietes by means of an escharotic, before making the opening ; in which case I can scarcely imagine that the operation would prove one either of the slightest difficulty or danger. But even supposing that it were not entirely free from either, it is still surely wiser to give the patient at least the chance of having his life prolonged, than to permit an inevitably fatal affection to run an uninterrupted course, which we know can, at best, be calculated by months only ? In my humble opinion, until the troubles of life become *permanently* greater to an individual than its pleasures, it is more philosophic for him to try and

bear 'the ills he has, than fly to others that he knows not of.'

From all that has been said it will appear that I do not regard the establishment of a biliary fistula in a human being as a formidable operation, and that, when once it has been successfully established, the benefits it would bestow upon the patient would be very considerable. For, in the first place, it would not put him in one whit worse a condition than a quadruped which has been in a similar way operated upon for experimental purposes; and the only bad symptoms, as we know, which are likely to arise from it are simply those resulting from the non-admittance of bile into the intestines. Which fact cannot, of course, be used as an argument against the operation, seeing that it is in those very cases where the disease itself has precluded the entrance of bile into the intestines, that the performance of the operation is advocated. So that if the artificial fistula does not diminish the whole of the evils, it will at least in no case increase even a single one of them.

The triumph of operative surgery would of course be to establish an artificial fistula between the gall-bladder and the duodenum. For then not alone would the evils resulting from the pent-up bile be removed, but those arising from the non-admittance of bile into the intestines likewise be at the same time overcome. I am not quite sure if, in these

days of antiseptic surgery, this operation is not practicable; for I can see no reason why the adjacent surfaces of the gall-bladder and duodenum should not be eroded by potassa fusa and speedily stitched together. Adhesions would rapidly form (see case reported at page 671, where eight hours sufficed to seal up the opening), and a permanent duodenal fistula be thereby established. My almost invariable complete success in all forms of abdominal operations on animals (gastric fistula, &c., &c.) have led me to the conclusion that the danger in the human subject arises almost solely from the operation being delayed until the patient's constitution is so broken down by the disease, that, when in the end it is undertaken, the poor sufferer has not strength left in him to bear it. Whereas, had he been operated upon before his constitution became broken down, the operation might, perhaps, have been as successful as if it had been performed on a healthy dog. The difference in the amount of danger between operations on healthy dogs and unhealthy men not being so much due to organic constitutional peculiarities, as to the differences in their vital powers, from the one being in robust health, and the other in an advanced state of disease, when operated upon. So, in advocating the artificial establishment of a biliary fistula, or the extraction of an impacted gall-stone from the common bile-duct, in a human

being, my advice is—operate early, instead of waiting until the patient has no vital stamina left in him.

In those cases of jaundice from permanent obstruction where it is considered unadvisable to adopt the plan of operating above suggested, we ought in our general treatment carefully to avoid the common error of administering foods likely to produce an increased secretion of bile. For the sufferings of the patient are not so much due to a deficient secretion, as to the want of biliary excretion. Our whole energies should therefore be directed to sustaining the strength of the patient, and mitigating, if possible, the pathological effects of the absence of bile from the intestines. This, I believe, we can best do by artificially supplying the place of the absent bile in the digestive process. *Not, however, in the way usually recommended, of giving inspissated, ox or sheep's, bile along with the food;* a method of treatment which originated ere modern physiology rent the veil of therapeutical empiricism. For, in the first place, all the bile prepared according to the methods indicated in the pharmacopœias, has its most essential properties destroyed during the process of preparation. In the second place, the only bile which closely approaches in its composition that of man, is that of the omnivorous domestic pig, whose food not only, but even whose digestive process, closely resembles that of the human being. And, in the third place,

we have hitherto been instructed to administer it at the very time which modern research has proved to be the most unsuitable that could possibly be devised. In administering bile immediately after food, as is usually done, we most effectually produce the contrary result to what is intended. For when bile mingles with gastric juice, it destroys its digestive power. So that, by giving the bile immediately, or soon after a meal, we really diminish instead of increase the power of the digestive functions. My experiments, both chemical and physiological, led me to propose, twenty years ago, not only another method of preparing bile for medicinal purposes, but also to suggest an entirely new mode of administering it.

As regards the method of preparation. Nothing can be more simple, and at the same time more effectual. Fresh bile, taken directly from the gall-bladder of the newly-killed pig, is filtered, through very porous filter-paper, to free it from mucus ; it is then as rapidly as possible evaporated to dryness at a temperature not exceeding 160° Fahr. The bile, as soon as dried, is ready for use. Simple as this operation appears in theory, there are two practical difficulties connected with it :—1st, Bile filters very slowly, and consequently little must be put into the filter at a time. 2nd, Bile is rather hygroscopic, and consequently, in order to get it dried quickly, it is

necessary to spread it over a large surface. If the bile has been well prepared, that is to say, thoroughly freed by filtration from its ferment mucus, and well dried, it will keep in stoppered bottles for many months, and in gelatin capsules for many years, without losing any of its active properties. I have at this moment in my possession some that were so prepared twenty-one years ago, and they are still, to all appearance, in a perfectly good condition.

Having stated that bile as at present employed more frequently does harm than good, by retarding instead of hastening the digestive process, I have now to point out the manner in which it may be given with advantage.

If bile be administered, as I propose, at the *end* of stomachal digestion, it will, as in the healthy organism, act on the chyme at the proper moment, and thereby render it fit for absorption. In order still further to ensure the action of the bile being delayed until the food is in a condition favourable to its action, that is to say, until it is ready to pass from the stomach into the duodenum, I had the bile, as above prepared, put into gelatin capsules, which are not readily acted on by the gastric juice. While in the stomach, the capsules, however, swell up from the size of a pea to that of a small gooseberry, and at the same time become so soft that they readily burst in passing through the constricted pylorus into the duodenum,

and thereby allow the bile to escape, and come in contact with the food at the precise moment its action becomes requisite in the digestive process.[1]

Each capsule should contain five grains of the prepared bile. Five grains being equal to about a hundred grains of liquid bile fresh from the gall-bladder. Two capsules therefore represent two hundred grains of pure bile, a quantity which, though perhaps less than the healthy organism consumes during each digestion, would in most cases be sufficient for the wants of the system. If, however, a larger amount be considered necessary, there is no reason why three or more capsules should not be given. By the administration of prepared bile in the manner here described, the physician is enabled to imitate nature, and supply an important element to the system; which, although incapable of curing the disease, can nevertheless aid in warding off for a time the fatal termination.

[1] Prepared bile, made up into an ordinary pill, dissolves in gastric juice in a quarter of an hour. When the pill is silvered it is dissolved in half an hour, and when gilded, in forty minutes. Whereas, in experiments made many years ago on the same specimen of gastric juice, the gelatin capsules prepared for me by Savory & Moore, although swollen to more than three times their original size, were nevertheless intact at the end of an hour and a half. They readily broke on being gently squeezed between the finger and thumb; it is not therefore probable that they would pass through the pylorus without giving way, and allowing their contents to escape. Capsules of pig's bile prepared as above may be had at the wholesale druggists', Burgoyne, Burbidges, & Co., 16 Coleman Street, E.C.

Sloughing, Gangrene, and Rupture of the Gall-bladder.

These conditions must be regarded as exceedingly rare, for one seldom reads in medical periodicals of their occurrence. Some cases of the kind I will, however, mention, each with special characters of its own, so as to present the reader with different types of these pathological conditions.

Gall-bladders have not only sloughed, but become gangrenous and ruptured, from the effects of gall-stones. At least, such was supposed to have been the cause of death by hæmorrhage in a patient seen by Dr. Leared, who in 1859 exhibited the parts at the Pathological Society.

The case was that of a man, aged 22, who, after suffering for a few days from agonising pain in the region of the gall-bladder and navel, suddenly fell into a state of collapse, and died within a few hours. On *post-mortem* examination the gall-bladder was found not only to be ruptured, but in a gangrenous sloughing condition, the rupture being caused, Dr. Leared thought, by over-distension, the only thing found to account for which was a small pea-sized calculus blocking up the duodenal orifice of the common bile-duct. The man was ill only a fortnight, and his symptoms commenced with paroxysmal pains—like gall-stone colic—in the right hypochondriac region, with tenderness on pressure over the

gall-bladder. These symptoms were associated with slight jaundice. As I, however, told Dr. Leared at the time, I had grave doubts of the correctness of his theory that the gangrenous condition and rupture of the gall-bladder was the result of its over-distension. The argument I advanced against the theory was that gall-bladders distended with pent-up bile to twenty times greater size neither become gangrenous nor rupture. Therefore I thought that some other cause induced the sloughing of the gall-bladder. There can, at the same time, be no doubt whatever that a rupture of the gall-bladder may arise from its over-distension in cases where there exists ulceration or other thinning and weakening disease of its coats, induced by the presence of gall-stones, &c. But when this kind of rupture, in contradistinction to an ordinary perforation, occurs, it is usually rapidly followed by symptoms of acute peritonitis. For further information on this subject see what I have said regarding the rupture of the common bile-duct in the chapter devoted to gall-stones, page 629.

Rupture of the gall-bladder has occurred during protracted labour; and undoubtedly, when such an untoward accident occurs, it must be attributed more to a diseased state of the gall-bladder itself than to the mere severity of the labour. As I know nothing of such cases personally, and none of them has been reported sufficiently fully or lucidly to enable me to

draw any conclusions as to their morbid anatomy, I decline to say anything more on the subject.

Carbonate of Lime Deposits—erroneously called Ossification—of the Gall-bladder.

The name of ossification of the gall-bladder has been, I believe, erroneously given to cases where an extensive deposit of amorphous carbonate of lime has taken place within the viscus, without a trace of true ossification having been detected, or even ever existed. Gall-bladders have not only been found lined with, but entirely filled up by, a deposit of carbonate of lime, exactly like what is sometimes met with in the cystic duct. Dr. Murchison recorded a case of this kind, where at the *post-mortem* no bile whatever was found in the gall-bladder. It was collapsed, and had its mucous membrane encrusted with a layer of white carbonate of lime, which effervesced strongly on the addition of nitric acid. The specimen was taken from the body of a woman aged 37, who died from cirrhosis of the liver, coupled with hypertrophied spleen and leukæmia. The gall-bladder end of the cystic duct was completely blocked up by the calcareous matter. (Pathological Society's 'Transactions,' vol. vii. p. 240.)

In 1850, Dr. Ogier Ward showed at the Pathological Society a portion of a gall-bladder (having small calcareous plates embedded in it), which was dis-

charged through a fistulous opening in the right iliac region of an unmarried lady aged 48. The history of the case was somewhat peculiar. She had suffered from purulent expectoration and signs of a cavity in the left lung for twenty years, and about eight years previous to the portion of the gall-bladder being discharged, she was attacked with diarrhœa, vomiting, and fainting, when a tumour was for the first time discovered in the right side, apparently unconnected with the liver, as it was more in the centre of the abdomen, and had a marked sulcus between it and the liver. In a twelvemonth's time it had descended into the right iliac fossa, and there it pointed and burst midway between the pubes and spine of the ilium. From it came a purulent hydatid discharge mixed up with whole hydatid cysts varying from the size of a pea to that of a turkey's egg. Some singly, some in clusters, and striated externally. In five weeks the hydatids ceased to be discharged, and the pus became less offensive. The fistula, however, did not heal up until it had been open for four months. When suddenly, after a bilious attack, the discharge ceased, and the wound closed. The right side of the abdomen at the same time becoming tense and tender, and the patient suffering from rigors. Four days later, the orifice reopened, and a quantity of pure bile, at the rate of about a pint a day, discharged itself for nine days, when a slough of a portion of the coats of

the gall-bladder came away. The bile continued to pass, in gradually diminishing quantity, until, by the end of seven months from the first bursting open of the fistula, only about a drachm of bile was discharged in the twenty-four hours. This was at the time the specimen was shown to the Society, and when the patient was still only in a very moderate state of health.

Although gall-bladders that are found completely filled with a deposit of lime are, I may say invariably, described as being cases of ossification, yet, when the deposit is chemically and microscopically examined, it is found neither in its composition nor its histology to bear any resemblance whatever to true ossification, no matter however hard and bone-like it may be. For it consists not only chiefly of carbonates instead of phosphates, but is structureless, being a mere heterogeneous mass. This pathological condition of gall-bladder is unfortunately one of those which furnish no specific signs or symptoms during the lifetime of the patient; so at present it is to us of no clinical importance whatever.

For some further information on the subject of calcareous deposits, see pages 584 and 605.

Cancerous and other Growths of the Gall-bladder.

Like the liver itself, the gall-bladder is liable to become affected with both malignant and benign

forms of tissue degeneration, and the exciting cause appears in many cases to be, as in the case of the liver, the irritative effects of gall-stones. Gall-stones apparently, indeed, not only give rise to serious organic changes in the walls of the viscus, such as inflammatory thickenings and hypertrophies ; but to the formation of new growths in the shape of benign scirrhous and malignant encephaloid tumours. (See chapter on Cancer.)

In most books these tissue changes in the coats of the gall-bladder are said to be merely secondary to liver-tissue degenerations ; but this, like many of the other old-fashioned notions which have been passed down from sire to scion, is only another of the erroneous notions arising from generalising upon imperfect data. For not only does recent pathology prove that most of the gall-bladder tissue changes occur independently of liver diseases ; but that they are themselves actually in many instances the exciting cause of secondary degenerations in the liver itself. In support of this opinion I quote the following series of cases illustrative of its correctness from different points of view.

The first case is one reported by Dr. Markham of a woman aged 28, who died in St. Mary's Hospital. The patient was intensely jaundiced, and her chief symptoms were pain and vomiting, coming on about a quarter of an hour after taking food. She died

apparently from exhaustion arising from the combined effects of inanition and pain. The urine was deeply bilious, while the stools never contained a trace of bile. The only physical sign detected was the existence of a hard swelling at the pyloric end of the stomach, which led to the erroneous diagnosis of pyloric disease.

At the *post-mortem* examination, the gall-bladder was found to be completely converted into a hard solid mass of scirrhus, which, Dr. Markham thought, had commenced in the coats of the viscus itself. It had thrown out at one or two points several processes into the liver tissue. The centre of the mass contained a number of gall-stones, which naturally originated the belief that the irritation caused by their presence excited the disease.

A case of cancer affecting the gall-bladder, where the presence of gall-stones also appears to have been the probable exciting cause, is related by Dr. Sidney Coupland in the thirty-first volume of the Pathological Society's 'Transactions,' as occurring in a woman aged 56. The tumour could be readily felt as a swelling just below the false ribs, and was associated with sharp pain in the right hypochondriac region. At the *post-mortem* no trace of a gall-bladder could be found, but occupying its place was an ovoidal epithelial cancerous mass, the size of a cocoa-nut; in the substance of which were found numerous small orange-coloured calculi of the

size of split-peas. And it appears highly probable that it was the irritative effects of these calculi in a predisposed constitution which called the cancer of the gall-bladder into existence. In support of this theory I may cite another case of supposed primary cancer of the gall-bladder associated with gall-stones. It is related in the same volume of the Pathological Society's 'Transactions' as the preceding, by Dr. Norman Moore. It occurred in a woman aged 59, and was felt as a hard tumour near the liver, and at the *post-mortem* the gall-bladder was found infiltrated and surrounded by a mass of medullary cancer, while its interior contained four large and several small gall-stones, which Dr. Moore, I think judiciously, remarks might be regarded as the exciting cause of the cancer.

Cancer of the Common Bile-duct.

Cancerous tumours of the common bile-duct are by no means rare, but they are seldom or never diagnosed during the lifetime of the patient, from the simple fact that their signs and symptoms possess nothing characteristic about them. I may mention that the jaundice produced by cancer of the bile-duct is as intense and as permanent as it can possibly be, and that it invariably produces all the worst collateral symptoms which the most inveterate impacted gall-stones can produce. An excellent illus-

tration of the truth of this remark is afforded by a case which Dr. Vanderbyl published in the ninth volume of the Pathological Society's 'Transactions,' page 230.

The case was that of a man, aged 36, who had been in good health up till within about six months of his death, when he was suddenly seized with vomiting and purging, and in a week or two afterwards became jaundiced, the stools pipeclay-coloured, and the urine loaded with bile. The liver being both enlarged and tender to pressure. After a month or two the liver got smaller in size, and the gall-bladder became perceptible to the touch. The jaundice got more and more intense. The patient became delirious, emaciated, with œdematous lower limbs, sank, and died. At the *post-mortem* the liver weighed sixty ounces. The gall-bladder measured, from entrance of duct to fundus, seven and a half inches, and contained fifteen ounces of a pale liquid, like barley-water, of a specific gravity of 1010, and alkaline reaction. All the biliary ducts, even those in the substance of the liver, were dilated by a similar kind of liquid. An obstruction was found at the duodenal orifice of the common bile-duct, caused by a medullary growth in its interior.

In concluding this chapter, my treatise on affections of the biliary organs might not inappropriately be brought to a close ; but, as a notorious punster,

on being asked if life was worth living for, wittily answered—'All depends upon the liver'—and every physiologist knows that this jocular reply embodies a great and philosophical truism. Not only from the fact that the functions of all animal organs are correlated *inter se*, and reciprocally dependent for their individual healthy activity one upon another, but from the equally important fact that from the functions of the liver being fourfold (see page 58), its normal action is *ipso facto* one of the most essential factors of healthy life, it may be well for me to add a supplementary chapter of hints on differential diagnosis, which will not only most probably be very acceptable to those who, from being actively engaged in practice, have but little time to devote to study, or even to keep themselves *au courant* with the rapid advance of medical literature, but besides may suffice to refresh the minds of those who have perused the book, with the facts that have been taught and exemplified in the text, showing that not alone the kidneys, the spleen, and the skin, but even the lungs, to a certain extent act vicariously with the liver. And, what is more, that the biliary function cannot get out of order without the whole nervous system becoming seriously deranged. The brain itself even becoming diseased. For not only do headaches and deliriums result, but even hallucinations are engendered, and actual in-

sanity sometimes follows in the wake of hepatic derangements. Indeed, the very name 'melancholia' is a compound of the two Greek words, 'black' and 'bile,' and, as every one knows, the sallow, bilious look of the melancholic monomaniac is something more than a mere proverbial phrase. Indeed, it is well known that not alone have threatened attacks of simple melancholia, but even of desponding religious mania, been occasionally staved off, if not actually cured, by a judicious and timely restoration of the normal functions of the liver.

It will, I think, be scarcely necessary for me to waste space by recapitulating all that has been said in different parts of the text regarding the reciprocal action of the liver and other organs; for every wary practitioner is supposed to have his eyes open to all forms of vicarious hepatic function, and never to fail, during the course of diseases affecting the general system, to keep careful watch upon the supervention of liver derangements. From it being a generally recognised fact that there is not a single febrile or non-febrile constitutional disease—not even gout, syphilis, or diabetes excepted—which does not demand, as an essential preliminary to convalescence, the more or less perfect restoration of the hepatic functions. The following hints will, however, be no doubt useful to all actively engaged in practice.

CHAPTER XXVII.

HINTS TO AID IN THE DIAGNOSIS AND PROGNOSIS OF DISEASES OF THE LIVER.

As upon correct diagnosis success in practice chiefly depends, this chapter will perhaps by some be regarded as not the least important in my book, from there being no doubt whatever that treatment is, comparatively speaking, easy when diagnosis is correct. And it may be truthfully said that a patient's life, and with it probably the well-being of a family, may entirely depend upon his medical attendant's ability to early recognise the true nature of the hepatic affection under which he labours.

As the hints now about to be given must of necessity, on account of space, be put forward without explanatory details, it is clear that they will be more particularly useful to those who have made themselves acquainted with the cases, facts, and theories given in the text; and therefore it may be as well for me to inform those of my readers who may feel disappointed at the absence of explanatory infor-

mation that they can themselves readily remedy this defect by turning to the chapter to which the individual hint specially refers, where they will find all the particulars connected with it given in abundant detail.

It may also, perhaps, be as well for me to inform the reader who has not already gleaned the fact from the text, that the writer, rightly or wrongly, implicitly believes in the truth of the subjoined clinical aphorisms :—

a. That science enables one to accomplish in the domain of practical medicine what empirical means totally fail to do.

b. That true science gives the clue not only to the shortest, but at the same time to the easiest, way of arriving at a correct diagnosis.

c. That while a knowledge of science reconciles us to the inevitable, it at the same time prevents us attempting the impossible, even in the domain of practical medicine.

d. That an apparently obscure case in the so-called 'practical diagnostic' sense may become as clear as noonday when the teachings of experimental physiology are brought to bear upon it.

e. That errors in diagnosis, leading to mistakes not alone in prognosis but in treatment, are frequently capable of immediate correction by

the application of a chemical test to some one or other of the excretions.

f. That moderate abilities, when aided by scientific knowledge, often succeed in unravelling the pathology of obscure hepatic diseases, where transcendental abilities, even when coupled with great experience, are found to be utterly helpless.

These being the views I entertain, the following are the main facts on which I rely.

1. Mostly all liver diseases are hereditary. Even many of those apparently accidentally acquired—such as gall-stones—are no exception to the rule.

2. Babes are liable to be attacked with precisely the same forms of liver disease as adults, even true malignant disease being no exception to this rule.

3. Several hepatic affections have a distinctly prenatal origin, not alone such as malformations of the bile-ducts, but even hydatids, syphilitic and cancerous growths, hepatitis, and biliary concretions.

4. Pain is a symptom common to almost all the varied forms of hepatic disease—at least at some part or another of their course. To wit, hepatitis, cirrhosis, inspissated bile, gall-stones, abscess, and cancer. Indeed the only generally

5. Painless affections are hydatids, embolisms, fatty and amyloid degenerations of the liver. But even

these may be associated with acute pain. As, for example—

a. When the hydatid cyst inflames or suppurates.

b. When hydatid vesicles block up the bile-ducts.

c. When fatty, amyloid, or syphilitic livers become attacked with hepatitis.

6. The KIND of the pain, as well as its accompaniments, is an important aid in making a differential diagnosis. For while in

a. Malignant disease the pain is of a dull continuous and decidedly subacute character, that from

b. Biliary concretions or entozoa blocking up the bile-ducts is acute and in general paroxysmal.

c. The pain of hepatitis is not only acute but continuous, and

d. When the capsule of the liver is implicated, especially the part covering the upper convex surface in contact with the diaphragm, an increase of pain accompanies each deep inspiration.

e. When suddenly developed excruciating hepatic pain exists without jaundice, it usually arises from a foreign body impacted in the cystic duct.

7. As there are no less than six causes of right hypochondriac pain which are liable to be mistaken for

that arising from the presence of a biliary concretion, before forming a diagnosis, founded chiefly upon the symptom of hepatic pain, even when it is of a distinctly colicky character, it is necessary to bear in mind the fact that it may not be due to a biliary concretion at all, but to—

a. A urinary calculus lodged in the infundibulum of the pelvis of the right kidney, or in the right ureter.
b. An attack of pleurodynia; or of
c. Pleurisy of the right side.
d. Acute gastritis.
e. Duodenitis; and
f. Partial peritonitis.

8. Shoulder pain is an unreliable symptom of any form of hepatic disease whatever.

9. There are hepatic enlargements both of a painful and a painless kind. Their differentiation may, in a measure, be assisted by a knowledge of the facts that—

a. The most rapid form of painless enlargement—occurring sometimes within a couple of months—is the fatty.
b. The painless amyloid kind is of much slower growth than the fatty, taking from six months to nearly as many years for marked development.

c. While a painless fatty liver seldom extends below the umbilicus, an amyloid one occasionally reaches as far down as the crest of the ilium.

d. The only other form of painless enlargement of the liver is that caused by a hydatid.

This last form of painless enlargement, from its being circumscribed, *ununiform*, and in general only implicating the lower edge of the liver, is less likely to be confounded with the above-named forms of hepatic painless enlargements than with four forms of disease yet to be alluded to.

10. The hydatid enlargement of the liver is usually at its lower margin. On account of the cyst always growing in the direction of the least resistance, it projects downwards into the free abdominal cavity. The hydatid enlargement is apt to be confounded with an ovarian tumour, a dilated stomach, a phantom tumour, pregnancy, and a distended gall-bladder.

The hydatid tumour may be differentiated from

a. AN OVARIAN TUMOUR, by not being movable along with the uterus, nor palpable to a vaginal examination.

b. A DILATED STOMACH, by not being associated with great gastric disturbance.

c. A PHANTOM TUMOUR, by not disappearing on

the patient being put under the influence of chloroform.

d. PREGNANCY, by the sound of a fœtal heart being detectible after the third month.

e. A GALL-BLADDER distended with bile, by the absence of jaundice and other signs of biliary derangement.

11. The painful forms of hepatic enlargements are all the acute and chronic varieties of hepatitis, biliary concretions, idiopathic and traumatic abscesses, and cancers. These may, in a measure, be differentiated by paying attention to the following facts—in addition to their ordinary characteristic symptoms :—

a. In simple hepatitis, acute or chronic, the liver seldom extends below the umbilicus ; while

b. In malignant disease it may reach the crest of the ilium, and has usually, at the same time, a nodulated surface.

c. All forms of hepatitis and suppurations are associated with more or less febrile disturbance.

d. Malignant disease is associated with a cancerous cachexia, and very often has

e. A cancerous family history, as well as

f. An absence of pyrexia.

g. The pain of cancer is continuous, though subacute.

h. That of biliary concretions is paroxysmal as well as acute.

12. Great enlargement of the liver, with subacute pain, in general proceeds from encephaloid disease.

13. An advanced and softened encephaloid of the liver communicates to the hand of the examiner an indistinct feeling of fluctuation, which, by the inexperienced, is apt to be attributed to suppuration. From suppuration it is easily differentiated by remembering that while along with a softened cancer there is sure to be a cancerous cachexia, along with suppuration of the hepatic parenchyma there is equally certain to be a rapid pulse, a furred tongue, and great febrile disturbance.

14. The only other form of hepatic disease a suppurating liver is at all likely to be confounded with is hydatid. From which the following characteristics are in general sufficient to differentiate it :—

a. The suppurating enlargement is painful on pressure.

b. Is accompanied by great constitutional disturbance.

c. The fluctuating part is usually surrounded by a resisting inflammatory ring.

d. An abscess never, by any chance, gives a vibratory thrill when percussed—the so-called hydatid fremitus—whereas a hydatid occasionally does.

e. A suppurating hydatid is tantamount to an abscess as regards its signs and treatment, and consequently has only a differential prognostic importance from its being much less fatal.

15. Whenever, in the course of acute or chronic hepatitis, RIGORS supervene without any more probable cause, the advent of suppuration may be diagnosed. For abscess of the liver is liable to occur in every chronic as well as acute form of hepatitis.

16. All chronic inflammatory painful enlargements of the liver are invariably followed by atrophy —if the patient survives sufficiently long for the pressure of the inflamed, and subsequently hypertrophied, connective tissue to act deleteriously on the secreting cells.

17. As jaundice is not only a common accompaniment of hepatic disease, but at the same time, as regards its modes of appearance and disappearance, an invaluable sign in forming a diagnosis and prognosis, as well as a useful guide in the selection of remedies, I shall here give some hints as to the differential diagnosis of its various forms and phases.

18. Although jaundice frequently proceeds from a suppression of the biliary function arising from all the various forms of hepatitis—idiopathic, malarial, toxic, and traumatic—the most common causes of a yellow condition of the skin are, in this country at least, those of the obstructive variety. For—

19. A yellow discoloration of the skin, with pipeclay-coloured stools and saffron-tinted urine, makes its appearance whenever an obstruction to the flow of bile into the intestines has existed for a few days—even occasionally for so short a time as seventy-two hours. Thus jaundice arises from :—A congenital deficiency of ducts, accidental obstruction in course of common bile-duct—inspissated bile, gall-stones, hydatids, and other forms of entozoa—closure of outlet of the common-bile duct from pressure of pregnant uterus, abdominal tumours, impacted fæces in transverse colon, disease of pancreas or of neighbouring organs, ulcer of duodenum, inflammatory stricture of common bile-duct.

20. In this country, and probably in all equally temperate as well as colder climates, jaundice, as the result of obstruction by biliary concretions, occurs, I believe, in at least 58 per cent. of all the cases of jaundice met with. Yet be it remembered that jaundice NEVER arises from the presence of gall-stones either in the gall-bladder or in the cystic duct.

21. Every case in which there is a history of a previous attack of temporary jaundice, and the patient complains of acute pain in the region of the liver of a paroxysmal character, may be put down as one of impacted biliary concretion. For in ninety out of every hundred of such cases this diagnosis is almost sure to be the correct one.

22. In every case of jaundice the result of obstruction caused by the presence in a duct of a biliary concretion, no matter whether it be inspissated bile or a true gall-stone, the jaundiced condition of the skin is preceded by and associated with pain.

23. Paroxysmal pain—hepatic colic, as it is commonly called—is never, or scarcely ever, absent when a gall-stone is doing sufficient mischief to be able to induce jaundice.

24. Gall-stones, and even large ones, may exist for years in the gall-bladder and other parts of the body of patients, not alone without producing jaundice, but without causing pain, at least sufficient pain to call for treatment. So that their presence may not even have been so much as suspected, and their existence be only accidentally discovered at the *post-mortem* of their proprietor.

25. Although, as soon as a concretion leaves the gall-bladder and becomes impacted in the cystic duct, acute pain, excruciating it may even be, immediately occurs, no jaundice supervenes. No sooner, however, does the stone reach the common bile-duct, where it prevents bile reaching the intestines, both from the gall-bladder and the hepatic duct, than jaundice makes its appearance. Moreover, I may further remark that while the stone is impacted in the cystic duct, although there is no jaundice, the pain is associated with quite as much sickness, febrile

disturbance, and nervous depression, as when marked jaundice is present.

26. A stone is known to have passed from the cystic (after having been impacted there) into the common bile-duct, by the sudden supervention—in addition to the paroxysmal pain and febrile disturbance—of pipeclay stools and high-coloured urine, along with a jaundiced condition of the skin.

27. After having said that a gall-stone may be impacted in a duct without inducing jaundice, I ought to add that I have never seen, heard, or read of a case of any biliary concretion whatever becoming impacted in a bile-duct without being associated with pain.

28. Immediately after a gall-stone passes out of the common bile-duct, the previously existing pipeclay-coloured stools vanish, and a dark, bilious-looking motion immediately takes their place. The skin and conjunctivæ, however, retain their jaundiced hue for some days.

29. Neither the presence of jaundice, pipeclay-coloured stools, saffron-tinted urine, nor the amount of pain, depends so much upon the number, the size, or the shape of the gall-stones as upon the actual position they occupy in the liver and its appendages.[1]

[1] That the above hints are abundantly necessary was strikingly displayed in the discussion which followed the reading of a paper on 'Choleo-

30. The dangers accruing from biliary concretions do not always terminate with their safe extrusion either from the gall-bladder or bile-ducts, for they sometimes adopt strange ways of quitting their birthplaces. For example :—

a. Gall-stones make abnormal outlets for themselves through the coats of the gall-bladder or bile-ducts. The pressure of the stones against the walls of the gall-bladder or ducts setting up a sufficient amount of inflammatory action to give rise to a perforating ulcer, and the artificial formation of a direct channel between the biliary appendages and the stomach, intestines, peritoneal cavity, or pelvis of the kidney, through which the stones pass.

b. When a stone passes into the stomach, it is in general ejected by vomiting; the ulceration heals up, and the patient gets well. There are, however, exceptions to this favourable termination. As, for example, was shown in the cited case where gastric juice escaped through the opening made by the stone, and digested the living liver (page 1040).

c. If the stone passes into the intestines, it is usually safely expelled along with the stools.

cystotomy,' at the Royal Medical and Chirurgical Society, November 10, 1879, when some of the speakers showed that they possessed not only very hazy notions regarding bile-duct obstructions, but even of the uses of a human gall-bladder! See report in journals.

Sometimes, however, the stone is so large that it sticks fast in the ileum, or lodges in the ileo-cæcal valve, and induces fatal peritonitis or ileus.

d. A stone, after having ulcerated its way into the intestines, may become encysted in a *cul-de-sac*, and give rise to no further trouble; its existence being only discovered after death.

e. When stones ulcerate their way directly from the gall-bladder into the intestines, the duodenum is in general the seat of the intestinal communication. Whereas, when a cancerous growth is the cause of the perforating ulceration between the gall-bladder and the intestines, the colon is the usual seat of the communication.

f. Sometimes stones ulcerate into a blood-vessel, and a fatal hæmorrhage is the immediate result.

g. Cases have occurred where a sudden and fatal collapse has supervened upon the transposition of a stone from the bile-duct into the peritoneum.

31. The dangers arising from gall-stones do not necessarily terminate even after their safe expulsion from the body. For :—

32. A fatal occlusion of the duodenal orifice of the ductus communis choledochus may occur after the safe passage of a gall-stone into the intestines and its equally safe extrusion from the body. This

fatal form of occlusion occurs when the irritation caused by the transit of the stone through the bile-duct has been sufficient to set up an adhesive inflammation, or to induce ulceration, during the cicatrisation of which a permanent occlusion in the channel or at the mouth of the duct takes place.

33. It is of the greatest importance to the patient that a permanent occlusion of the common bile-duct —either from adhesive inflammation of its internal walls, a stricture, or the cicatrisation of an ulcer at its orifice—should not be confounded with a persistent jaundice from an impacted gall-stone, as in the two sets of cases not only is the prognosis, but the treatment entirely different. For so long as the jaundice is due to the presence of an impacted gall-stone, there is hope of a perfect recovery taking place. Whereas when the jaundice is due to occlusion of the channel of the duct, either by inflammatory adhesion of its walls, a stricture, or by the cicatrisation of an ulcer at its orifice, a fatal issue is inevitable.

34. Although it is comparatively easy to diagnose the supervention of a permanent organic occlusion of the bile-duct after a gall-stone is known to have passed through it, it is quite another matter to attempt to differentiate between an occlusion arising from a simple stricture of the bile-duct and one arising from the cicatrisation of an ulcer at its orifice, except in one particular class of cases. For example :—

a. Whenever, after the extrusion of a gall-stone, the disappearance of jaundice is only temporary, and the skin slowly, gradually, and *painlessly* resumes a well-marked and permanent icteric tint, in nine out of every ten of such cases the return of the jaundice is not due to another gall-stone blocking up the duct, but to the duct becoming permanently and inevitably fatally closed up, either by a constriction or an adhesive inflammation of its internal walls, or by the cicatrisation of an ulcer at its duodenal orifice.

b. When an ulcer at the duodenal orifice of the bile-duct cicatrises, it almost invariably includes in its cicatrix the orifice of the pancreatic duct. So that when this double occlusion occurs, the differential diagnosis is simplified by the fact that not only are the stools pipeclay-coloured, but when cold covered over with a layer of fatty matter. And when cod-liver oil is administered to the patient by the mouth, the surface of the fæces assumes the appearance of 'Windsor soap.' (See page 771.)

35. From a naked-eye examination of an extruded gall-stone, useful information may be gleaned. Even its shape alone tells us much. Thus, for example, if it possesses many facets, it has not been a solitary occupant of the gall-bladder or bile-ducts, but is one

of a multitude, and, if at the same time of small size, it may be one of a dozen, or of a dozen hundreds. If it has only two or three facets upon it, there are not likely to be many more stones left behind. If it is circular or oblong, and has no facets, although the presumption is that it is a solitary stone, this is by no means invariably the case ; for I once, as previously said, saw four gall-stones, as large as small hazel-nuts, pass within a few days from a patient, all of which were globular, and without a single facet upon them.

36. A concretion of inspissated bile is distinguished from a gall-stone by assuming a purple-red tint when, after being crushed into powder, strong sulphuric acid is poured over it.

37. When in any case of jaundice there is a sudden cessation of the pipeclay-coloured stools, the cause of the jaundice is almost sure to have been one of two things—a biliary concretion or an entozoon, blocking up the hepatic or the common bile duct.

38. The obstruction to the outflow of bile caused by entozoa may not only occur from—

a. A hydatid tumour closing up the channel of the common bile-duct by external pressure ; but from

b. The parasite growing in or penetrating into its interior and blocking it up.

c. Even cases have been recorded where an obstruction to the common bile-duct has been occasioned by the presence of liver flukes (*Distoma hepaticum*) in the common duct. Also

d. Others, in which the obstruction of the duct is said to have arisen from the entrance into it of worms from the intestines. Cases of this kind are exceedingly rare, as well as difficult of diagnosis, from the symptoms they present being in no way characteristic, except when a large hydatid is the obstructing cause. In which case its diagnosis is aided by a knowledge that the obstructive jaundice was preceded by a painless tumour, and the supposition is of course reduced to an axiomatic certainty when cyst vesicles are found in the fæces.

39. The supervention of jaundice arising from an obstruction within the duct caused by entozoa, although much slower than that arising from gall-stones, is nevertheless more rapid than when the obstructing cause is the slow development of a morbid growth or a cicatrising duodenal ulcer.

40. When jaundice supervenes *suddenly*, without pain, and there is no other assignable cause, the probability is that it is due to the pressure of a hydatid tumour on the duct.

41. Cases of jaundice arising from obstruction caused by entozoa, either in the form of hydatids or

worms, are always perplexing in their diagnosis, for the following reasons :—

a. When the parasite is impacted within the bile-duct, it produces precisely the same symptoms and signs as a biliary concretion does when it is impacted in a similar situation, even to the symptoms of paroxysmal colic and itching of the skin.

b. The signs and symptoms of both impacted entozoa and gall-stones may suddenly disappear, and not a vestige of either be detected in the patient's stools. So that both the existence of the signs and the symptoms, and their mode of disappearance, may be equally well accounted for on the supposition that they were due to the presence of a living animal parasite or of a dead crystalline concretion.

c. If, however, any other part of the patient's body be infested with hydatids, the diagnostic scale is to be turned in that direction. But in all other cases, when there is no proof of the patient being the subject of hydatid disease, it is wise policy to give the benefit of the doubt in favour of a biliary concretion being the *fons et origo* of the jaundice, for the simple reason that while cases of jaundice induced by entozoa are exceedingly rare, those arising from biliary concretions are proportionately common. Hence

I may say that there are ninety-nine chances out of every hundred that if a biliary concretion is blamed for doing the mischief the diagnosis is likely to be correct.

42. Jaundice of a well-marked and permanent character may not only arise from malignant and other tumours affecting the secreting substance of the liver itself, but from precisely the same forms of growths attacking the stomach, intestines, pancreas, the left kidney, and supra-renal body, through the tumour inducing a stoppage to the flow of bile into the intestines from causing its occlusion by external pressure on its walls. In the majority of these cases the differential diagnosis between the jaundice arising from gall-stones and morbid growths is, comparatively speaking, easy enough ; from the fact that in the latter set of cases the jaundiced condition of the skin comes on very slowly, and is in most of the cases preceded by the usual train of symptoms denoting the presence of cancerous disease in the other organ or organs affected. For example, if the disease has commenced in the stomach, duodenum, kidney, or pancreas, it will have been preceded by the characteristic symptoms of disease in these parts.

43. Occlusion of the bile-duct, besides occasionally arising from the implication or external pressure of cancerous and other growths beginning in the hepatic parenchyma and neighbouring organs, may

also arise from a cancerous or other pathological form of tumour primarily developing itself within the common bile-duct.

44. There is as yet no known way of making a differential diagnosis between jaundice arising from the external pressure of a tumour on the bile-duct, and that produced by the formation of a growth, either primarily or otherwise, in its interior. Fortunately, however, for both doctor and patient, the treatment to be followed is in both sets of cases the same, so that the being able to make a differential diagnosis in these cases is of no importance in a clinical point of view. The line of treatment being simply to alleviate distressing symptoms and retard as far as possible the inevitable fatal result.

45. The disease most commonly mistaken for cancer of the liver is gall-stone impacted in the common bile-duct, especially when it is ulcerating its way into the intestines.

46. Another form of disease which occasionally, though rarely, produces jaundice from obstruction to the outflow of bile through the common duct, is enlargement of the glands in the portal fissure. Although the cases recorded are but few in number, I observe that almost every one of them has been noted as being associated with ascites.

47. Yet another and almost equally rare source of jaundice is that caused by the presence of fæces

impacted in the flexure of the colon, which, fortunately, is of easy diagnosis from its being associated with all the usual symptoms of intestinal obstruction.

48. A case of obstructive jaundice the result of an aneurismal tumour is reported to have been attributed to gall-stones, from its pulsations having been mistaken for the normal throbbing of the abdominal aorta communicated through the tumour to the hand of the examiner. An error which I think might possibly have been avoided by the employment of the stethoscope; for, to the trained ear, a true aneurismal is quite different from a normal aortic murmur.

49. The presence of bile acids in the urine, in any case of jaundice whatever, is crucial proof that there is reabsorption of secreted bile going on; and the knowledge of this fact is such an important diagnostic agent in all obscure cases, that whenever the rough and ready method of testing the urine for bile acids does not yield clear evidence of their presence or absence, the urine ought to be subjected to a proper and complete chemical analysis.

50. It must never be forgotten, in differentiating cases of obstructive jaundice, that :—

a. The suppressive form of jaundice occurs in all cases of hepatitis. No matter what the cause of the hepatitis may be.

b. That in most cases of atrophy, when jaundice is present, it is due to suppression. Notwith-

standing that the fæces may be of a dark, tarry, bilious hue. For in such cases the dark colour of the stools is, as in contagious jaundice (yellow fever) and acute atrophy, not due to the presence of bile, but of blood.

c. After the jaundice has been ascertained to be due to obstruction, the cause of the obstruction may be suspected to be :—

d. Gall-stones, if it be associated with paroxysmal pain, and itching of the skin.

e. Cancer, if the pain be a mere dull ache, the organ enlarged, and a cancerous cachexia present.

f. A stricture or ulcerative occlusion of the common bile-duct, induced by the passage of a gall-stone, if, after colicky pains and the temporary disappearance of the jaundice, the skin gradually and slowly resumes the icteric tint.

g. Cirrhosis, or tumour, aneurismal or other, implicating the veins, if there be ascites.

h. While if there be evidence of a hydatid or any of the other tabulated diseases which produce obstructive jaundice, the cause of the occlusion of the duct may be, in the absence of other evidence to the contrary, attributed to it.

i. When the jaundice is the result of pressure upon the common bile-duct from cancerous tumours or enlarged glands in the portal fis-

sure, there is usually enlargement of the superficial abdominal veins, as well as more or less ascites.

j. When jaundice supervenes very slowly, and is unattended with pain, its cause is probably due to the existence of a cicatrising ulcer or to a morbid growth.

k. It is to be remembered, however, that no matter whether the case be a complicated or a simple one, if jaundice exists with it at all, and is not the result of suppression, unless there be a *complete* obstruction to the outflow of bile from the common bile-duct, it can never be severe.

l. Itching of the skin in a case of jaundice is in all cases, in my opinion, a pathognomonic sign of obstruction.

m. In cases of cancer of the liver, the daily amount of uric acid excreted is often higher than, but seldom below, the normal standard.

n. Hydatids, especially the multilocular variety, have been occasionally erroneously diagnosed as cancer.

o. So also has amyloid degeneration of the liver.

p. In almost every case where jaundice supervenes within four days after an attack of colic, the cause is the impaction of a gall-stone in the common bile-duct.

q. There are four sometimes equally acute, and in general fatal, forms of jaundice, which so closely resemble each other in their symptoms as to be frequently mistaken for each other; but the mentioning of their names will be sufficient to guard the reader, when the histories of the cases are known to him, from confounding them with each other. They are the more virulent forms of—

(1) Acute atrophy of the liver.
(2) Contagious jaundice.
(3) The jaundice from mental emotion, and
(4) The jaundice from poisons.

51. As a knowledge of the proportional frequency with which jaundice is met with in different forms of disease is of great service in making a differential diagnosis, I subjoin my views of the matter as follows :—

a. Jaundice occurs in about 99 per cent. of the cases where a gall-stone is in the common bile-duct.

b. Jaundice occurs in about 90 per cent. of the cases of chronic atrophy of the liver.

c. Jaundice (in a greater or less degree) occurs in about 70 per cent. of cases of hepatic abscess.

d. Jaundice (at least in a modified form) is met with in about 30 per cent. of the cases of congested liver.

e. Jaundice only occurs in 6 per cent. of the cases of cancer of the liver.

f. Jaundice occurs in about 2 per cent. of the cases of hydatids of the liver.

g. Jaundice (that is to say, marked discoloration) only occurs in 1 per cent. of the cases of amyloid and fatty degeneration of the liver, though a sallowness of the complexion is present in nearly all of them.

52. While the presence of jaundice is a pathognomonic sign of hepatic derangement, its *entire absence* is no proof whatever of the non-existence of even grave hepatic disease.

53. Many fatal forms of hepatic disease are frequently unattended with either yellow skin, pipeclay-coloured stools, or saffron-tinted urine. Such may be the case in cancer, abscess, amyloid, syphilitic, and other hopelessly incurable degenerations of the parenchyma of the liver.

54. When jaundice occurs in the course of any disease, be it hepatic or other, it either arises from a suppression of the biliary secretion, or from an obstruction to the outflow of the secreted bile.

55. Jaundice arising from suppression is almost always—I might even go so far as to say invariably—attended with more or less grave constitutional symptoms.

a. In that arising from enervation, although the

attack may be exceedingly sudden, it is, from its very onset, accompanied by cerebral disorder.

b. Those forms of jaundice depending on acute or subacute atrophy of the liver, though little less sudden in their onset, are always equally attended with cerebral disorder.

c. Jaundice arising from blood-poisoning, whether it be from an animal, vegetable, or mineral poison, is invariably associated with marked febrile symptoms.

d. Jaundice from hepatitis in any form is invariably accompanied by more or less acute constitutional inflammatory symptoms.

e. Dark stools in cases of jaundice from suppression, even when there co-exists bile-coloured urine, is indicative of the presence of BLOOD, not of bile, in the fæces.

56. Variations in the intensity of the discoloration of the skin are met with in almost all forms of permanent jaundice, possibly from the elimination of the pigment through the skin proceeding more rapidly at one time than at another.

57. Be it remembered that the depth of the jaundiced tint of the skin is more proportionate to the duration of the disease than to its pathological cause.

58. When ascites is present in a case of liver disease, it is in all cases the direct result of a mechanical

interruption to the flow of the venous blood out of the abdomen, caused by pressure on the vena cava inferior, either by a tumour, or by a contracted condition of the liver itself.

59. Œdema of the lower limbs, as well as enlargement of the abdominal veins, is always absent in uncomplicated cases of abscess, hydatids, gall-stones, cancer, fatty, amyloid, and other forms of parenchymatous degeneration of the liver. When there is an exception to this rule, it probably arises from the constricting effects of the external pressure on the ascending cava.

60. It is well always to remember that the palpable existence of one form of disease of the liver does not necessarily preclude the co-existence of another. Thus gall-stones, for example, may be associated with almost any form of hepatic affection, hydatids, cancerous tumours, fatty livers, &c. So again a hydatid may be associated with a cancer, a cancer with cirrhosis, syphilitic gummata with hepatitis, and so on through the whole round of diseases.

61. The presence of blood in the stools, either in the form of pure blood, grumous matter, or coffee-grounds, is in hepatic cases not necessarily the result of active hæmorrhage. It may equally be the direct result of a passive congestion of the portal veins, or rectal hæmorrhoidal vessels, caused by atrophy or any other form of hepatic disease inducing venous obstruction.

62. The right lobe of the liver is more prone to be affected with disease than the left; and that too not with one specific form of disease only, but with several—differing widely in their pathological nature —to wit, abscess, cancer, and hydatids.

63. In cases of tumour of the liver, when there exists a doubt as to its true nature, the exploring needle ought to be had recourse to, as the fluid or solid withdrawn immediately reveals its probable nature. Thus, should it be a hydatid, the liquid is of a specific gravity of from 1007 to 1012, limpid, loaded with chlorides, and not coagulable by heat. If ascitic or ovarian, it is of a specific gravity of above 1012, and coagulable by heat and nitric acid. Should the liquid be from a hydronephrosis, it contains (like true urine) both urea and chlorides, but not necessarily albumen. If again the tumour chance to be an abscess, the liquid withdrawn is purulent; but it must be borne in mind that hydatids suppurate, and gall-bladders are occasionally filled with pus. While, lastly, if pure bile comes away, the case is one either of distended bile-duct or gall-bladder. For further information refer to the chapter on the exploration of the liver.

Additional Hints to aid in forming a Prognosis.

64. In no case of hepatic disease whatever can the amount of danger to life be estimated either by

the depth of the jaundice or by the intensity of the pain. For even in the case of gall-stones inducing a fatal perforation, jaundice may be totally absent, and in the case of inevitably fatal occlusion of the common bile-duct by the cicatrisation of an ulcer, there may be scarcely any pain complained of by the patient.

65. A patient who has once suffered from an attack of jaundice from biliary concretions, unless careful prophylactic treatment be adopted, is almost certain to be affected with it again.

66. A hydatid tumour once emptied never refills.

67. Although cases of suppurating hydatids are dangerous, they are by no means always fatal.

68. Patients labouring under malignant disease of the liver usually die within a year after the appearance of the cancerous cachexia.

69. When jaundice has become so persistent and intense as to leave no doubt of its being due to a permanent obstruction to the outflow of bile into the intestines, the patient may in general be expected to succumb, from the deleterious action of the pent-up bile—even under the most favourable collateral circumstances—within two or two and a half years from its commencement,[1] and within six or ten

[1] At page 16 of the second edition (1821) of his work on disorders of the liver, Dr. Ayre says:—'About a year ago, a gentleman of this town died, who, during eight years, was jaundiced from a permanent obstruction existing in the biliary duct. During the whole of this time

months after the hepatic parenchyma has begun to shrink and atrophy.

70. When, after a long-continued jaundice (associated with dull aching pain towards the latter period of its duration) the stools become of a natural colour, and then slowly resume a pipeclay appearance, the patient is likely to die within six or ten months after the temporary improvement has ceased, as it is most probable that the cause of the jaundice was at first a gall-stone impacted in the common bile-duct, which ulcerated its way out of it. While the resumption of the pipeclay stools is in its turn due to an occlusion of the duct, either by stricture, adhesive inflammatory action, or the cicatrisation of an intestinal ulcer induced by the perforating stone.

71. In cases of jaundice from obstruction, with intermissions, there is no immediate danger to life; for so long as the gall-bladder can occasionally, even partially, empty itself, or bile flow directly from the liver into the intestines (as it is oftentimes able to do, even in severe cases of obstruction from impacted

the bowels were often excessively and variously disordered; yet at no period, as I learnt from him—and as I had often occasion to observe myself—were the discharges from the bowels black.' This eight years' case of jaundice from obstruction in no way militates against what I said regarding the patient's usually succumbing to the deleterious effects of the pent-up bile within two years after *complete* stoppage to its flow into the intestines; for in Dr. Ayre's case there is nothing to show that the obstruction was associated either with a distended gall-bladder or bile-ducts, and consequent backward pressure on the secreting structure of the liver. (See remarks in paragraph 71.)

gall-stones, in consequence of the stone accidentally changing its position and allowing the bile to flow past it), the patient (other things being favourable) may live almost to his natural term of life. I have known cases go on for six or more years, and, as I previously said, I have a certain Lady N—— now (1882) under treatment who has been intensely jaundiced for seven years, from impacted gall-stone, and who, though still dark in colour, is otherwise nevertheless both in good health and spirits.

72. Acute abscess of the liver, occurring in temperate, is not less dangerous than in hot climates. Unless opened early (that is to say, before the hectic stage has begun), a fatal issue, in the majority of instances, may be foreboded.

73. Chronic abscess of the hepatic tissue may exist for years, may open spontaneously into the intestines, then either heal up entirely, or reform and again burst into the bowels, lungs, or elsewhere; in the end killing the patient, either suddenly, or slowly by hectic and exhaustion.

74. Acute, or, as it is sometimes called, yellow, or malignant, atrophy is by no means the inevitably fatal disease it is in general pictured to be, for under a judicious system of treatment many cases recover.

75. The appearance of tyrosin and leucin in the urine in the course of any hepatic disease may be

regarded as indicative of atrophy of the liver—acute, subacute, or chronic.

76. When, after the administration to the patient of benzoic acid or the benzoate of soda, hippuric acid appears in the urine, as it invariably does under normal circumstances, it may be taken for granted that a part of the hepatic tissue is still performing its proper functions ; for when the hepatic functions are entirely arrested this chemical transformation ceases.

77. When sugar appears in the urine in a case of persistent jaundice, a fatal termination may be suspected of not being very far off.

78. The supervention of cerebral symptoms in the course of jaundice—either of a delirious, convulsive, or comatose character—is always a sign of danger.

Finally. Although sociologists tell us that it is a thankless task to proffer unasked-for advice, I shall on the present occasion lay aside all fear of the unasked-for advice I am about to offer being either received with ingratitude or contempt, from the simple fact that it is only intended for those of my professional brethren who are just entering on the threshold of practice, and if it does them no good it will assuredly do them no harm. It is this :—

Always begin the investigation of a case of hepatic disease by listening attentively to the patient's

own ideas of its nature and origin. For, practical medicine, as I said before, being simply common sense on a scientific foundation, and all patients being neither hypochondriacs nor fools, very often a clue to the pathology of the case may be directly gleaned from the patient's own lips.

Never interrupt a patient during the narration of his symptoms, unless you have a particularly clear-headed one to deal with, or you will almost for a certainty set his ideas off at a tangent, and, like a clock that is being continually wound up, his narrative will never cease.

In gauging the value of symptoms, remember that it does not entirely depend upon their particular nature, but also potently on the mental capacity, physical courage, and veracity of the patient who describes them.

Never pooh-pooh a patient's ideas either of the cause or nature of his case. For some grave hepatic diseases have strangely trifling exciting causes; as for example, mental emotion, a blow, an indigestion, and such like.

Do not begin the physical until you have completely exhausted your verbal examination of the case.

When drawing conclusions from the result of a physical examination, bear in mind that, just as the value of symptoms depends on the intelligence and

veracity of their narrator, in like manner does the intrinsic value of physical signs depend on the manual skill and medical knowledge of the examiner. So much so, indeed, is this the case, that an accomplished examiner will elicit valuable diagnostic data in an obscure case, where an untutored one, even though, perchance, more highly mentally gifted, will fail to elicit anything likely to lead to a correct diagnosis.

Never until after having carefully considered the symptoms described by the patient along with the visible signs and personally elicited data in connection with the historical career of the illness, venture to give an opinion as to the nature of the disease.

(I think that this last piece of advice will scarcely be considered superfluous by any one who has perused the seventy-eight hints I have tabulated as aids in the differential diagnosis of hepatic diseases.)

Having made up your mind that you thoroughly understand the pathology of the case, and can give to it an appropriate name, then unhesitatingly state, either to the patient himself or to his friends, as may appear under the special circumstances to be best, the opinion you have arrived at. But when in doubt, equally frankly and unreservedly say that you desire to consult with a professional brother, before venturing to express a decided opinion as to the nature of the case. For depend upon it, in pro-

fessional as in commercial matters, honesty is always in the long run the best policy. In fact, my experience of consulting practice has given me ample opportunity of noting that it is only the dishonest and ignorant who ever decline to take counsel with their medical brethren.

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